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PROCEEDINGS

OF THE

TWENTY-EIGHTH GENERAL MEETING

OF THE

NATIONAL VETERINARY ASSOCIATION.

HELD AT

THE HOTEL CECIL, LONDON,

ON

July 27th and 28th, 1910.

H. & W. Brown, Printers, 20 Fulham Road, London, S.W.

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National Veterinary Association.

GENERAL MEETING,
1910.

President

Professor James Macqueen

Life Vice-Presidents

Principal McCall
Mr. J. Malcolm
J. F. Simpson, J.P.
J. Mackinder
J. M. Parker
E. Faulkner
W. Hunting
W. Bower
A. W. Mason

Mr. W. H. Bloye
Matthew Hedley
Prin. J. R. U. Dewar
Sir J. M‘Fadyean
Mr. F. W. Garnett, J.P.
Chas. Allen
R. C. Trigger, J.P.
W. Shipley
R. Roberts

Vice-Presidents

Mr. T. S. Price
G. Bowman
C. Sheather

Mr. S. Stockman
S. Villar
F. L. Gooch

Council

Prof. H. A. Woodruff
Mr. A. L. Butters
S. H. Slocock
F. G. Samson
Dr. J. McI. McCall
Prof. G. H. Wooldridge

Mr. W. J. Mulvey
Henry Gray
J. B. Martin
A. Wilson
J. Willett
F. Hobday

Members of the Association who are also President or Secretary of an existing local Veterinary Society—

Presidents :

Mr. H. G. Bowes
W. Burt, Junr.
Dr. O. Charnock Bradley
Mr. J. Holland
P. J. Howard
T. Eaton-Jones
T. Salusbury Price
W. Woods

Secretaries :

Mr. W. Ascott
W. Awde
J. Clarkson
H. J. Dawes
A. Gofton
G. H. Locke
H. A. MacCormack
G. P. Male.
J. Robertson
J. Alex. Todd

Provisional Committee

Chairman : Mr. T. Salusbury Price.

Mr. C. Roberts
F. Willett
H. Simpson
W. Burt, Junr.
E. Batt
H. W. Caton
Alfred Broad
A. Rogerson
P. Simpson
H. King
H. A. MacCormack

Mr. W. K. Stuart
J. A. Gosling
W. Perryman
D. Pugh
R. C. Tennant
W. A. Hancock
W. J. Mulvey
J. Alex. Todd
C. Pack
E. H. Leach
Prof. E. S. Shave

Hon. Secretary : Mr. W. L. Harrison

Trustees

Messrs. R. C. Trigger ; J. F. Simpson, J.P. ; F. W. Garnett

Auditors

Mr. S. Villar and a professional accountant.

Treasurer (*pro tem.*)

G. H. Wooldridge, 30 Brixton Hill, London, S.W.

Secretary

Wm. Hunting, 5 Halkin Place, Belgrave Square, London, S.W.

SECRETARY'S REPORT.

The Twenty-Seventh Annual Meeting was held at Harrogate on July 7th and 8th, 1909, under the Presidency of G. Bowman, Esq., M.R.C.V.S.

The proceedings were published and issued to all the members.

A Council Meeting was held at 10 Red Lion Square, on November 10th, 1909. at which the Provisional Committee was elected.

A Council Meeting was held on February 9th, at 10 Red Lion Square, at which delegates were appointed to the Health and Sanitary Congress as under:

Professor Wooldridge and Mr. S. Villar to the Health Congress at Birkenhead in July.

Professor Macqueen and Mr. R. Roberts to the Sanitary Congress at Brighton in September.

The prolonged illness of Mr. Jas. F. Simpson forced the Council to appoint Prof. Wooldridge to act as interim Treasurer.

The Committee appointed at Harrogate to formulate a scheme for the affiliation of all Veterinary Societies with the N.V.A, has occupied much time throughout the year. The final meeting of the Committee was held at Crewe, on May 7th, when the suggestions made by different Societies were considered and as far as possible embodied in the scheme. Copies of the new proposals have been sent to each member of the N.V.A., and the scheme will be brought before the members on the first day of the meeting this year in London.

The Provisional Committee have made all arrangements for the Annual General Meeting to take place at the Hotel Cecil, London, on July 27, 28, and drawn up the following order of proceedings :

THE NATIONAL VETERINARY ASSOCIATION.

Dr. *Treasurer's Cash Account from 18th June, 1909, to 17th June, 1910.* Cr.

	£	s.	d.
To Balance at Bank at 18th June, 1909	245	8	4
,, Subscriptions received between June 18th, 1909 and 17th June, 1910	155	19	0
,, Dividend on £400 Consols four quarters to 5th April, 1910	9	8	4
	£410	15	8

	£	s.	d.	£	s.	d.
By Expenses of Annual General Meeting at Harrogate, viz :						
Banquet Expenses ...	20	0	0			
General Expenses ...	31	0	11			
Reporting Proceedings ...	28	4	0			
				79	4	11
,, Printing, Publishing Proceedings of Meeting, Postage and Advertising				68	13	8
,, Delegates' Expenses at the Hague Conference and the Leeds Congress ..				25	4	0
,, Sundry General Expenses, viz :						
Treasurer's Payments for Printing, Stationery and Postage	3	15	0			
Accountant's Fee for preparing Accounts ...	2	2	0			
Hire of Room (R.C.V.S.)	1	1	0			
				6	18	0
,, Balance at Bank				230	15	1
				£410	15	8

I have prepared the above Account from the books and information submitted to me and hereby certify the same to be in accordance therewith. I have examined the receipts and securities of the National Veterinary Association and found them to be correct, and certify that the balance at the Bank on June 17th, 1910, was £230 15s. 1d., and that the amount of £400 is invested in Consols in the name of the appointed Trustees.

A. D. Wykes, *Chartered Accountant*, 50 Cannon Street, E.C.

Sidney Villar, F.R.C.V.S., Amersham, Bucks.

21st June, 1910.

National Veterinary Association.

Meeting at the Hotel Cecil, Strand, London, W.C.,
On July 27th and 28th.

President: Professor J. MACQUEEN, F.R.C.V.S.

ORDER OF PROCEEDINGS:

FIRST DAY (Wednesday, July 27th).

10 a.m. Minutes and Correspondence: Report of Delegates to the Hague Congress: To arrange place of meeting: Election of President and officers for ensuing year: Consideration of Affiliation Scheme.

"Shivering and Stringhalt"
By Prof. John R. McCall, M.R.C.V.S.

Discussion opened by Prof. G. H. Wooldridge, F.R.C.V.S.

1.30 p.m. Luncheon

2.30 p.m. **"Contagious Granular Vaginitis in Cattle,"**
By Ainsworth Wilson, F.R.C.V.S.

Discussion opened by S. Stockman, M.R.C.V.S.

7 p.m. for **7.30** **Annual Banquet**

SECOND DAY (Thursday, July 28th).

10 a.m. **"Strangles"**
By Capt. A. G. Todd, M.R.C.V.S.

Discussion opened by Sir John M'Fadyean, M.B., B.Sc., C.M., LL.D., M.R.C.V.S.

1.30 p.m. Luncheon.

2.30 p.m. **"Rickets in the Dog"**
By F. W. Cousens, M.R.C.V.S.

Discussion opened by H. Gray, M.R.C.V.S.

THIRD DAY (Friday, July 29th)

Luncheon and Tea at the Japan-British Exhibition, on invitation of the Central Veterinary Society and neighbouring ptactitioners,

TWENTY-EIGHTH GENERAL MEETING

Held at the Hotel Cecil, London,
July 27th and 28th, 1910.

Professor J. MACQUEEN, President, in the Chair.

Members present: Messrs. N. Almond, A. H. Archer, G. A. Banham, G. F. Banham, F. W. Barling, A. E. Bayley, A. J. Beckett, G. J. Bell, J. J. Bell, G. Bishop, G. Bloxsome, O. Charnock Bradley, A. L. Butters, J. Cameron, W. S. Carless, J. H. Carter, W. Caudwell, W. R. Clarke, P. W. Creagh, E. G. Crowhurst, D. G. Davies, H. J. Dawes, A. J. Dobbyn, J. B. Dunlop, A. Ellison, A. Gofton, C. H. Golledge, W. W. Grasby, H. Gray, R. W. Hall, E. R. Harding, W. L. Harrison, T. G. Heatley, M. Hedley, E. Wallis Hoare, F. Hobday, C. W. Howard, P. J. Howard, W. Hunting, R. Hughes, W. R. Jermyn, T. Eaton Jones, E. H. Leach, H. G. Lepper, A. Levie, G. H. Livesey, G. C. Lowe, H. A. MacCormack, Sir J. M'Fadyean, Messrs. S. J. Motton, J. H. Norris, C. Pack, J. H. Parker, C. F. Parsons, E. Price, T. Salusbury Price, D. Pugh, H. S. Reynolds, R. Roberts, T. A. Rudkin, F. G. Samson, C. Sheather, W. Shipley, P. J Simpson, S. H. Slocock, S. Smith, Lieut. J. J. M. Soutar, Messrs. A. Spreull, E. H. Stent, H. Sumner, F. B. O. Taylor, J. Temple, J. Thomson, Capt. A. G. Todd, Messrs. Sidney Villar, W. P. Weston, F. W. Willett, J. Willett, W. R. Williams, A. Wilson, W. Wilson, W. Woods, G. H. Wooldridge.

Visitors: Messrs. H. Begg, Col. L. J. Blenkinsop, W. H. Brown, Miss V. Cameron, Mrs. J. H. Carter, H. W. Caton, W. Golledge, Mrs. E. Wallis Hoare, J. W. Hopkin, M. D. Kerr, Miss M. B. McGavin, Lt.-Col. J. Moore, W. Perryman, Mrs. F. G. Samson, C. H. Sheather, Mrs. Spreull, Mrs. E. E. Stent, Mrs. W. R. Williams, Mrs. G. Wooldridge.

First Day, Wednesday, July 27th, 1910.

The President: The first part of our business this morning is connected with the Minutes and Correspondence. The Minutes of the previous meeting held at Harrogate have been already published and circulated to the members, and the Secretary informs me that there is no important correspondence that need be read. To be perfectly in order it will be necessary to have some member propose that the Minutes be accepted.

Mr. T. S. PRICE: I move that the Minutes be taken as read and adopted.

Sir JOHN M'FADYEAN: I second the motion.

The motion was carried.

The PRESIDENT: I have now the pleasure of calling on Dr. Bradley to read the Report relating to the National Veterinary Congress at the Hague.

REPORT OF DELEGATES TO THE INTERNATIONAL VETERINARY CONGRESS AT THE HAGUE.

As the representatives of the National Veterinary Association at the Ninth International Veterinary Congress held at The Hague in September, 1909, we have to report as follows:

The Congress was declared open on September 13th, by H.R.H. Prince Henry of the Netherlands, Duke of Mecklenburg, patron of the Congress. His Excellency the Minister of Agriculture, Industry and Commerce welcomed the delegates to Holland.

The President of the Congress, Professor W. C. Schimmel, of Utrecht, in an interesting speech, sketched the institution of International Veterinary Congresses by Professor John Gamgee, of Edinburgh. The first Congress held in Hamburgh in 1863 was attended by 102 veterinary surgeons, mostly German. Subsequent Congresses have been increasingly attended as the following figures show:—

Congress.	Year.	Place of Meeting.	Attendance.
1st	1863	Hamburg	102
2nd	1865	Vienna	163
3rd	1867	Zurich	188
4th	1883	Brussels	310
5th	1889	Paris	635
6th	1895	Berne	670
7th	1899	Baden-Baden	958
8th	1905	Budapest	1400

Speaking on behalf of the British Government, Sir John M'Fadyean said it was pleasing to note that veterinary surgeons in this country were beginning to take a greater interest in International Congresses founded by a countryman of their own. At The Hague Congress there were more representatives from the United Kingdom than had attended any of the previous conferences.

The subjects discussed by the Congress were both numerous and of the utmost importance. That the results of the discussions were of the greatest value was admitted by all, and that the Congress was a conspicuous success is beyond question.

Government efforts against Swine Fever and Swine Plague formed the subject of papers by Dr. M. Dorset (Washington), Dr. F. Hutyra (Budapest), Dr. R. Ostertag (Berlin), and Mr. Stewart Stockman. The last named reporter asserted that outbreaks of swine fever in England could be divided into three groups. (1) Virulent outbreaks characterised at the start by a short period of illness and a large number of deaths. The typical lesions of swine fever may be absent in such outbreaks. The virulence tends to die down, and the outbreak assumes medium virulence. (2) Outbreaks of medium virulence. The disease is more chronic and ulceration and diphtheresis in the alimentary tract are more frequent. (3) Still less virulent outbreaks. There are very few deaths, and the only evidence of something being wrong may be a general appearance of unthriftiness in a number of pigs.

The conclusions arrived at after the discussions were that Swine Fever and Contagious Pneumonia of Swine are two distinct diseases requiring different methods of treatment at the hands of the veterinary police. Swine Fever is due to a filterable virus, and ought to be eradicated by all possible means because of the great loss it causes to agriculture. In those districts where the disease is not firmly established, all diseased or suspected animals should be destroyed ; the owners being suitably compensated. In badly infected districts, the disease should be limited in its spread by putting into vigorous operation the customary measures directed against contagious diseases. Preventive inoculation can diminish mortality, and its investigation should be encouraged by Governments.

Less vigorous measures will suffice in dealing with contagious pneumonia of swine. Infected districts should be isolated, and the registration and control of trade in pigs should be rendered obligatory.

Naturally, the eradication of tuberculosis formed one of the most important matters for discussion. In connection with the subject of Governmental efforts against tuberculosis, with regard to the channels of infection, Professor Bang emphasised the fact that the spread of tuberculosis among calves and pigs largely depends upon the milk they drink. In minimising the spread by this channel the Danish laws have rendered inestimable service. According to the law of Denmark, all skimmed milk, butter, &c. destined as food for animals must be raised to a temperature of 80C.

The sanitary control of milk and the obligatory systematic inspection of meat supplied the basis for an important series of contributions by six reporters representing four nations—Germany, France, America and Great Britain. Consequently the views expressed may be taken as indicating expert opinion of the greater part of the civilised world. Mr. A. M. Trotter (Glasgow) did good service in again pointing out the crying need for uniformity in the matter of meat inspection. He urged the necessity for the formation of an international code by which shall be prescribed the method of inspection and the standard of adjudication to be adopted by the authorities of every country from which meat is exported. Moreover the Government of every country should insist on imported meat having been inspected according to the code. The working of the code should be under the control of a competent Government staff composed of veterinary surgeons, chemists and other officials.

In the control of our milk supply, Mr. Trotter airs a long-familiar grievance, when he says, "the medical officer of health claims to be the sole directing official on whom should devolve the responsibility of supervising the milk supply in all its complex ramifications"; and once more insists that every effort must be exerted "to ensure that the supervision of the health of the animals will be entrusted to veterinarians."

In view of the recent disclosures in connection with the meat-packing industry of his country, it was interesting to hear certain parts of the report of Dr. A. D. Melvin, Chief of the Bureau of Animal Industry at Washington. He said, "The law of June 30th, 1906, absolutely prohibits the use of all preservatives or colouring matters which are unwholesome. The Secretary of Agriculture in carrying out this law has declared that the only preservatives which may be used on meats or meat food products for inter-State trade are common salt, sugar, wood-smoke, pure spices and saltpetre. With regard to shipment to foreign countries, the regulations provide that upon written direction of the foreign purchaser preservatives may be used which do not conflict with the laws of the country to which the particular meat food products are to be shipped." Thus, said Dr. Melvin, it is plain "that the United States Government is not only endeavouring to protect its own citizens by every legitimate means, but that we are also doing everything in our power to uphold the laws of the countries to which our meat food products are shipped.

The United States Government authorities have drawn up a list of rules for the guidance of inspectors when dealing with tuberculous carcases. These seem to supply a starting point in the adjustment of the international code which Mr. Trotter suggests should be instituted.

How carcases and meat should be treated with the object of rendering them harmless, formed the subject of a report of some moment, drawn up by Dr. F. von Puntigam, Municipal Veterinary Inspector at Brunn (Moravia). This reporter recommended the inspection by Government veterinary officials, of flocks and herds prior to slaughter, the inspection of carcases, and the confiscation of any part or parts held to be unfit for human food. In case the veterinary inspector considers that by sterilization any portion or portions of the carcases can be rendered fit for human food, he shall have power to order such treatment. Dr. von Puntigam's recommendations met with general acceptance and were adopted by the meeting.

The relationship of Avian Tuberculosis to Tuberculosis of Mammals has formed the basis of research by bacteriologists for many years; but even now the question is not settled to universal satisfaction. Dr. S. Arloing (Lyons) was the chief sphaker on this subject at one of the general meetings of the Congress. His conclusions are of interest. He regards avian tuberculosis as being essentially the same as tuberculosis of mammals. Its bacillus is merely a variation of the bacillus of Koch, and is itself subject to variations in the same way as is the human or bovine bacillus. Clearly, therefore, the same precautions should be taken in dealing with avian tuberculosis as with tuberculous meat.

In view of the present importance, and the promise of future extensive application, the study of protozoa in relation to disease naturally contributed to the discussions of the Congress. Also, quite naturally, veterinary surgeons from this side of the North Sea were particularly interested in what was said by Dr. A. Theiler, Veterinary Bacteriologist to the Transvaal Government. Dr. Theiler pointed out that a fly and an animal formed the two necessary links in the chain required for the propagation of a protozoan " tropical disease." In order, therefore, to produce the disappearance of the disease it is clearly necessary to break or unlink the chain. Prophylaxis of fly or mosquito-borne disease, consequently, has two objects:—

(1) To save the greatest number of exposed susceptible animals by rendering them immune, and also by fly-proof seclusion.

(2) The eradication of the disease by destroying the reservoir or the host.

It is interesting to note that Dr. Theiler is convinced that piroplasmosis of sheep does not exist in South Africa. Records of its occurrence, he asserted, depended on an error of diagnosis.

After the discussion the meeting unanimously agreed that Governments which have not as yet organised a veterinary service should have impressed upon them the necessity of doing so as soon as possible. Wherever possible, scientific veterinary investigators should be sent into those territories in which are prevalent diseases not yet investigated. An International Bureau of Tropical Diseases of Animals, consisting of veterinary representatives from all countries concerned, should publish a bulletin containing the results of all recent international publications.

Whether the preparation and the control of serums and bacterial products should rest with the State or with the individuals gave rise to an animated discussion. Speakers supported State control: other speakers were of opinion that only individual control was feasible. At the close of the discussion it was resolved that, "In default of direct preparation by the State of products of a bacterial nature for the use of the veterinary profession, the preparation and the sale of these products ought to be placed under the control of the State."

Among the many papers and demonstrations of the Congress, the memory of which will long remain with those who attend them, one of outstanding interest, and one greatly appreciated, was a lantern demonstration on the etiology and pathogeny of malignant new growths by Dr. E. F. Bashford, Director of the Imperial Cancer Research Fund. Dr. Bashford paid a graceful tribute to pioneer work done by veterinarians, and to the discovery by them of cancer in animals while prominent members of the medical profession were yet asserting that the disease occurred only in man. Moreover, the experimental study of cancer, he said, will ever be associated with the name Jensen, a veterinary surgeon.

A matter of some moment to the profession in this country was discussed under the title of "The conditions neceesary to obtain the Doctorate in Veterinary Science." On the Continent no less than in this country, opinion is prevalent that the degree of Doctor in Veterinary Science should be within the reach of those who desire to possess it. But on the Continent, as in this country, opinion is divided as to the conditions under which the degree should be granted.

It is clearly impossible for your representatives to give a detailed report of all the numerous papers which were submitted at the general meetings and the meetings of the five sections of the Congress. It is merely possible to single out such of the subjects of discussion as appear to be of most general interest. That all the papers were of value was admitted by everyone; and that the members of the Congress were materially benefitted by the discussions was questioned by none.

The social side of the Congress was not neglected; receptions, banquets, and excursions being certainly not less numerous than is customary on such occasions. An excursion which blended pleasure and profit was to Rotterdam, where a party was conducted over the Serum Institute by Dr. Poels, who, along with his assistants, explained the purposes and working of the Institute.

The grateful thanks of the British members of the Congress are due to all those who worked so strenuously to make the meeting the conspicuous success it was.

The next International Veterinary Congress is to be held in London in 1913; and it will behove the veterinary profession in this country to see to it that the London Congress does not fall short of its predecessors. Further, it is earnestly hoped that the British Government will take example from the action of other Governments towards past Congresses, and will grant such a sum of money as may allow of the reception of our foreign guests in a manner befitting the position our country holds in the scientific and veterinary world.

The PRESIDENT: I have now to call upon the Secretary to inform us with regard to the invitations received as to the place of our next meeting. Two invitations have been received, and our Secretary will tell you the decision of the Council last night.

Mr. W. HUNTING: The minute of the Council Meeting is as follows: "Invitations were read, one inviting the National Veterinary Association to hold its next Meeting in Scotland and the other to hold it in North Wales. It was decided to recommend the General Meeting to accept the invitation to North Wales."

The PRESIDENT: If there is no objection to the recommendation of the Council that we should accept the invitation to go to North Wales next year, I ask you to indicate your approval in the usual way.

The recommendation was unanimously adopted.

ELECTION OF OFFICERS.

The PRESIDENT: In connection with the Meeting next year the next point is to select a President, and after due consideration the Council last night decided to recommend to the General Meeting that Mr. William Woods, of Wigan, be our President for the ensuing year (Hear, hear).

Mr. J. J. BELL: I have great pleasure in moving that the recommendation of the Council be adopted. I think we could not have a better and more suitable gentleman as our President on the occasion when the Society meets next year.

Mr. T. S. PRICE: I have much pleasure in seconding that.

The motion was carried with acclamation.

Mr. WOODS: You are asking me to ride at a pretty stiff fence in accepting the Presidency at a meeting which is to be held a hundred miles away from my residence and in a district where I am unacquainted with any veterinary surgeons; but if it is the wish of this meeting, all that I can say is that I will do my best, and I can say no more (Cheers).

The PRESIDENT: The next point is the recommendation of the Council as to six Vice-Presidents, and the Secretary will give you the names recommended by the Council.

Mr. HUNTING: Mr. R. Hughes, Mr. E. Jones, Mr. T. S. Price, Dr. O. C. Bradley, Mr. Davis, and Mr. J. McGavin.

Mr. VILLAR: I have pleasure in proposing that these gentlemen be elected as Vice-Presidents.

Mr. R. ROBERTS: I have great pleasure in seconding that.

The motion was carried unanimously.

The PRESIDENT: The next business is the election of a Council for which we require twelve members, and Mr. Hunting will read the names of those recommended by the Council at their meeting last night.

Mr. HUNTING: Mr. H. Sumner, Mr. S. Jackson, Mr. E. Faulkner, Mr. Wynn Lloyd, Mr. J. B. Wolstenholme, Mr. G. H. Locke, Mr. R. Jones, Mr. F. W. Barling, Mr. W. A. Taylor, Mr. F. G. Edwards, Mr. J. H. Carter, Mr. D. Storrar.

The PRESIDENT: You have heard the names, and I shall be glad if someone will move that those be elected, if there are no additions to be made to the list.

Mr. J. MCCALL: I have much pleasure in moving that those names be accepted as the members of Council for the ensuing year.

Mr. F. G. SAMSON: I have pleasure in seconding that motion.

The resolution was carried unanimously.

The PRESIDENT: The next office is a very important one. Owing to very unfortunate circumstances it has been filled during a portion of the past year by Prof. Wooldridge, and at the meeting of the Council last night we sounded Prof. Wooldridge on his willingness to continue in office, and he very kindly offered to continue in office for another year. Therefore, if you agree with the recommendation of the Council that Prof. Wooldridge be asked to continue his appointment as Hon. Treasurer, I will ask someone to move to that effect.

Mr. J. CAMERON: I beg to propose that Prof. Wooldridge be appointed Treasurer for the ensuing year.

Mr. J. H. CARTER: I have pleasure in seconding that.

The resolution was carried unanimously.

The PRESIDENT: The next business is the Secretary. This is a very difficult question, but after due consideration the Council last night decided that they might continue to try the services of our friend Mr. Hunting (Laughter and hear, hear). He offered no objection, I believe, so long as we did not treat him too unkindly or lower his salary, which is not a princely one at the present time, but in view of altered circumstances it might be improved (Laughter). If any gentleman who has confidence in Mr. Hunting will move his re-election I shall be very glad to accept it.

Mr. G. A. BANHAM: I have very great pleasure in proposing the re-election of Mr. Hunting, on one consideration, and that is that he will look more carefully into the names of our members in connection with the list, and also that the offices which those gentlemen have filled shall be mentioned in future Reports.

Mr. W. WOODS: I should like to have the pleasure of seconding that motion. One of the great reasons that induces me to accept the Presidency is the fact that Mr. Hunting told me he would do the whole of the work, a work he can do very well, as we know (Hear, hear).

The PRESIDENT: I have no doubt that Mr. Hunting will pay attention to the condition mentioned by Mr. Banham, and that the Report next year will be quite a perfect one.

The resolution was carried with acclamation.

Mr. HUNTING: I think I ought to be allowed to accept the Secretaryship with a proviso to the effect that Mr. Banham will kindly let me have his corrected proof to put those numbers right (Laughter).

The PRESIDENT: The next part of the proceedings is to elect the late President, Mr. Bowman, of Leeds, as a Life Vice-President. That is a custom that has been pursued in the Association from the very earliest times, and I see no reason why we should not continue it. In the case of Mr. Bowman I think it is very well deserved, and I have very great pleasure myself in moving that Mr. Bowman, our late President, be elected a Life Vice-President of this Association.

Prof. WOOLDRIDGE: I have great pleasure in seconding the recommendation.

The motion was carried unanimously.

Mr. S. VILLAR: I rise to propose that this meeting, as representing the National Veterinary Association, do accord a most hearty vote of thanks to Mr. James Simpson for the services which he has rendered to this Association (Hear, hear). We heard with regret last night that it had become necessary to elect a successor to him both as Honorary Treasurer of this Society and as one of its Trustees. In both of those capacities, as well as in the capacity of an ordinary member, he has rendered great service to this Association, as indeed he has to the whole of the veterinary profession (Hear, hear). But mingled with our regret we were glad to hear last night that there was some recovery in his health and we hope that he will so far recover as to be able to appreciate—as I am sure he will appreciate—the hearty thanks of this Association and to know that we appreciate very much the good services which he has rendered to us (Cheers).

Prof. Wooldridge: May I be allowed to second that, and in doing so refer just for one moment to the very excellent condition in which the books of the Society and the accounts were when I was called upon, at an awkward moment, to undertake the duties that Mr. Simpson could no longer carry out for the time being. If one had not known Mr. Simpson before, one would have been afforded a very excellent opportunity of forming an opinion as to the very thorough manner in which he carried out the duties on behalf of this Association. In fact, it was no trouble whatever to take over the books, and to carry the work on exactly at the point where he left off, everything was in such good, ship-shape order (Hear, hear).

The resolution was carried unanimously.

The President: I take it you desire the resolution be inscribed upon the minutes and an intimation sent to Mrs. Simpson and the family. (Agreed to.)

Mr. P. J. Howard: I thought it would be hardly fair that this meeting of the National should pass over without passing a vote of condolence with the family of a very esteemed member of the Association, the late Mr. W. A. Byrne. I am sure it does not need any words from me to recommend the motion to you that a vote of condolence be sent by the Secretary of the Association to the late Mr. W. A. Byrne's friends in Ireland. His death is a great loss to the profession both in Ireland and England, and especially to this Association.

Mr. C. Roberts: I rise to second that proposal. It was with great regret that we heard of the death of Mr. Byrne. We all miss the cheery face and the kind words of our friend, and we are all exceedingly sorry that he has gone.

The President: I think we are quite agreed that this vote should be carried, and I will ask you to carry it standing.

The motion was passed by the members standing.

The President: The next item on the programme for to-day is the consideration of the Affiliation Scheme. Last night the Council considered this question at very great length, and formulated a resolution which the Secretary will now read to you. When you have heard this resolution, if you do not agree with it, it will be no bar to further consideration. At the same time there is a well-founded desire that there should be no waste of time over this question if you agree with the terms of the resolution. The resolution was passed by the Council last night and is submitted to-day for your acceptance.

Mr. Hunting: The following is the resolution: "That the Council, having considered the Affiliation Scheme, regret that they are unable to recommend its acceptance in its present form. They would, however, heartily approve of any workable scheme which would place the National Veterinary Association in closer relation with the local Societies, and they accordingly recommend that a Committee be appointed to consider the matter further and to report to the next Annual Meeting."

Sir JOHN M'FADYEAN: I beg to move that the recommendation of the Council be adopted by this meeting.

Mr. C. ROBERTS: I second that motion.

The PRESIDENT: The matter is now open for discussion, and anyone who desires to take a contrary course will have to move an amendment.

Mr. J. CAMERON: No one can approve more highly than I do of the principle of amalgamation, and I am sure we might consent to this resolution and see what can be done to get some benefits out of all our Societies. I am very pleased to find that some aspects of it were approved, but I was sorry to see there were other aspects which were not quite regular. Under the circumstances, I think the best thing will be to start anew, and I certainly approve of the lines laid down in the resolution.

The PRESIDENT: If there is no further criticism or suggestions I shall be obliged to put the resolution to the meeting.

The resolution was carried unanimously.

The PRESIDENT: Arising out of this resolution, it was considered by the Council that it would be advisable to appoint a Committee to carry out the reeommendation, and names were submitted last night and approved, and Mr. Hunting will now read them to you.

Mr. HUNTING: The names are: Messrs. J. Dunstan, W. Ascott, W. Woods, H. A. MacCormack, W. Hunting, J. A. Jordan, J. Weir, J. Macqueen, D. G. Davies, J. A. Todd, J. McKenny, H. G. Bowes, R. Hughes, W. Shipley, J. Clarkson, S. Villar, H. A. Woodruff, G. H. Wooldridge, Sir John M'Fadyean, Dr. O. Charnock Bradley as Chairman, and Prof. A. Gofton as Secretary.

Mr. HEDLEY: I propose that the recommendation of the Council be adopted.

Mr. T. S. PRICE seconded the motion, which was carried unanimously.

The PRESIDENT: In connection with this question a vote of thanks was passed by the Council, and it appears on the Minutes as a recommendation, that the Chairman and Secretary of the Committee—I refer to Dr. Bradley and Prof. Gofton—be accorded a vote of thanks for their services in connection with the Affiliation Scheme.

Mr. J. J. BELL: I have much pleasure in moving that.

Sir JOHN M'FADYEAN: I have much pleasure in seconding it. I am sure we all came somewhat reluctantly to the conclusion in the Council Meeting last night that we could not recommend this scheme in its present form, but that did not prevent us from recognising at the same time that we owed a debt of gratitude to the Committee, and in particular to Dr. Bradley and Prof. Gofton, for the great amount of time and trouble they have expended in drafting the scheme and generally in bringing it before the profession.

The resolution was carried unanimously.

Dr. O. C. BRADLEY: Might I on my own behalf and on behalf of Prof. Gofton express our appreciation of your vote of thanks. Naturally, we regret that the scheme has not come to greater maturity with greater rapidity, but there is considerable hopefulness, and we feel assured—and I am positive that everyone else who has considered the matter also feels assured—in a very short time a workable scheme, such as has been suggested by the motion will be put before this Association (Hear, hear). I thank you in the name of myself and Prof. Gofton for your vote of thanks.

"STRINGHALT" AND "SHIVERING."

By John R. McCall, M.R.C.V.S.,
Glasgow Veterinary College.

The importance of these conditions and the scarcity of veterinary literature bearing upon them indicates that the time has come when an opportunity such as that presented by the Annual Meeting of our National Veterinary Association may with advantage be employed in a free discussion upon these obscure but highly interesting diseases. In this way, no doubt, many interesting observations may be recorded, and new facts brought to light which will assist us all in arriving at more definite conclusions than apparently exist at the present moment.

Stringhalt is the term used to define that peculiarity—spasmodic and excessively rapid flexion of one or both hind limbs, which occurs when the horse is made to move, best seen in the slower paces, and particularly in turning or backing. The limb is lifted abnormally high, but is able to support weight and allow of other movement in the ordinary way. This spastic movement is most marked when the animal is first exercised, and if this be continued the symptoms become modified, thus suggesting that a degree of accommodation becomes gradually established between the flexors and extensors of the limb.

The clinical history shows that the disease usually becomes manifest when the horse is put to work, it increases with age and hard labour, and any circumstance which tends to lower the vitality of the subject undoubtedly aggravates the symptoms, e.g. an attack of influenza may make manifest a stringhalt hitherto unrecognised, which may, however, gradually disappear as the patient recovers strength. Excitement will also increase the symptoms, an example of which is frequently furnished in the Hackney Show ring. On the other hand, rest from hard labour has a modifying action, slight cases being difficult to detect after the animal has been laid off work for a few days.

The diagnosis of stringhalt in the early stages is usually an extremely difficult matter ; we may suspect the horse, and subject him to the most rigorous tests with negative results, yet, probably, were we to have the opportunity of examining him the next day we might have little difficulty in finding that he was affected. This intermittent nature of the malady is important, and should not be lost sight of in law cases where, naturally, the veterinarian is inclined to assume that because he is unable to detect any symptom of the disease it is consequently non-existent.

If the condition is at all marked, then walking the animal and suddenly turning him to one side and then the other, or moving him backwards and forwards, as a rule causes the characteristic movements; likewise if the horse is made to move over suddenly in the stall he may "pick his hocks." If in doubt subject him to these tests on cobbled stones, because in my experience a slippery surface exaggerates the tendency, and more particularly when the horse is yoked. This applies equally to shivering.

There can be little doubt that stringhalt is most prevalent among heavy draft horses, although we encounter it in the hackney, thoroughbred and other varieties; it also appears to be fairly common in the ass. At first sight its prevalence in draft horses might suggest that excessive muscular exertion played an important part in its production, especially when we remember that rest frequently modifies the complaint, but if excessive muscular exertion were the cause of the disease it would be of much more common occurrence. Admitedly muscular exertion *intensifies* the malady, but probably its effect is limited to this.

The immediate cause of this interference with movement has been sought for in different organs, but up to the present has not been clearly demonstrated. Falke identified the condition with sciatica in man, but the clinical history is absolutely distinct, sciatica being a painful affection, the attitude being due to restriction of movement on account of pain.

Renner took it to be due to chronic inflammation of the ischiatic nerve, but this cannot be accepted, as chronic inflammation of nerves leads to structural alterations in their texture, and causes muscular alterations in their texture, and causes muscular atrophy, whereas in stringhalt, if muscular atrophy ever exists, except as a general emaciation, it is extremely rare, and moreover, the microscopical examination of the ischiatic nerve fails to disclose structural alterations.

Boccar sought the cause in contraction of the peroneus, after discovering that the condition disappeared on section of this tendon; but this does not stand investigation, for section of the peroneus tendon in many cases is of little or no avail, and even in those cases where it is beneficial it is frequently several weeks after the operation before full results are obtained; this would not be the case were the condition due to such a mechanical cause.

Bone spavin has been included in the list of causes of stringhalt, and while it is possible to have bony ankylosis of the tarsal joint interfering mechanically with the flexion and giving rise to a peculiar form of false stringhalt, this is obviously not the cause of true stringhalt, because in the vast majority of cases of bone spavin there is no stringhalt, and in nearly all the cases of stringhalt which I have examined no evidence of bone spavin could be detected.

Bassi attributes true stringhalt to interference with the movements of the patella. We certainly meet with cases of this description, but the peculiar jerky catch here observed occurs at regular intervals, and is frequently accompanied by a clicking sound, which differentiates this from true spastic stringhalt where the movement is more rapid and rhythmic.

Contraction of the fasciæ of the thigh, or the tensor fasciæ latæ, has been credited with causing stringhalt, but when section of the peroneus tendon fails to produce beneficial results, section of the fasciæ also fails, and those few who have practised this surgical treatment have been unable to report favourably upon it.

Stringhalt has also been identified with chorea minor of man, and with locomotor ataxy. It certainly resembles the former closely in the clonic nature of the spasms, but differs in that stringhalt is *strictly localised* to a certain group of muscles, whereas in human chorea the spastic condition is more generalized—the arms, limbs, and facial muscles frequently being involved, especially under excitement or observation. Stringhalt certainly differs from human locomotor ataxy, not being so much a want of co-ordination and irregularity in muscular movement as a clonic spasm of certain muscles; blindfolding the animal has not, in my experience exaggerated the symptoms, nor interfered with the balancing power as it constantly does in locomotor ataxy of man. Furthermore, in the human subject sclerosis of the superior column of the spinal cord (*i.e.*, sensory) is practically constant, whereas this is not so in stringhalt of the horse. Another diagnostic symptom of locomotor ataxy of man is the want of light reflexes, but although I have applied this test to many animals affected with stringhalt, I could never say that any marked departure from the normal could be observed, although the iris of the horse appears normally to respond much more slowly to the light than is the case in the human subject.

In a pamphlet on "Diseases of the Nervous System in Horses," by Chalmers Watson, M.B., M.R.C.P.E., publishe a few years ago, the author gives a brief description of the condition known in the human subject as lateral sclerosis or spastic paraplegia To quote, "The clinical features are mainly motor, and comprise muscular weakness and a marked degree of spasticity in the gait. The affected limbs are raised in a spasmodic manner, and put down on the ground with a jerk that is often very striking and not unlike the type of movement seen in aggravated cases of stringhalt in horses."

Certainly the clinical picture presents a remarkable similarity, but in spastic paraplegia the lateral columns of the spinal cord are invariably affected, and the lesion is a gross one, this has not been found to be the case in stringhalt in the horse.

The symptoms exhibited in true stringhalt indicate that the cause is due to irregularities in the functions of the antagonistic nerves of the lumbar and pelvic plexuses, but whether these irregularities are functional or dependent on structural changes has still to be elucidated.

Hereditary Aspect of the Disease.

Whatever difference of opinion may exist as to the etiology of this affection, the tendency for animals affected with stringhalt to transmit the disease to their offspring has been so long recognised by veterinarians that it is needless to enlarge upon it. Most of us whose vocation brings us in touch with breeding stock have seen many examples, and quite a number present today could mention cases of stallions well known in the show rings of Great Britain, who are affected with this malady, and whose progeny are notorious for the same defect. It does not always follow that the disease is transmitted provided the dam is free from the defect, but still this is frequently observed. Where both parents are affected the hereditary tendency naturally becomes much aggravated, and this is common knowledge with breeders of horses. It is not usual for foals or yearlings to show the complaint, still such cases have come under my observation, particularly in hackneys. This hereditary character of the disease is quite in keeping with allied nervous affections in the human family, and there can be little doubt that the hereditary transmission of stringhalt plays a most important part in the perpetuation of the malady.

When we consider to what an extent in-breeding is practised in most of our pedigreed stock, this aspect of the subject assumes even greater significance; breeding with a mare so affected is bad enough, but the results are confined to her limited offspring, whereas in the case of the stallion, the effect is much more serious and far-reaching. Is it not time that the veterinary profession were bringing this fact prominently before the notice of the various Agricultural and Horse-Breeding Societies, and impressing upon these bodies the urgent necessity for examination of stallions destined for public service throughout the country? Already a few of the more enlightened and influential societies have grasped the situation and acted accordingly, and in the face of powerful opposition, but their example will require to become much more general before lasting benefit will result.

Legal Aspect.

In the case of Anderton *v.* Wright reported in *The Veterinarian*, July, 1871, after hearing evidence the magistrate, Judge Greene, stated "it was perfectly clear that stringhalt constituted unsoundness," and found for the plaintiff with costs. In the case of Thompson *v.* Pattison, tried before Mr. Justice Cresswell, at the Liverpool Summer Assizes, 1846, his Lordship said "it is a question for the jury whether stringhalt produces those effects which in the eye of the law render him unsound," and in summing up shortly afterwards his Lordship said to the jury, "you have heard the evidence as to stringhalt; if you are satisfied that it is a disease calculated to impair the natural usefulness of the horse you must find for the plaintiff, it being admitted that the horse had it." The jury found for the plaintiff.

Results of Stringhalt.

As previously stated this malady usually becomes worse with age and hard work, and few, if any, natural recoveries are recorded, still the extent to which it advances varies considerably, some animals, especially of the harness variety, appear to remain much the same for years, provided they are well fed and not overworked. The lorry horse who has to pull heavy loads on slippery streets with steep inclines yields to the malady much more quickly, and although in its initial stages the disease has little effect on his working capacity, the time comes when it undoubtedly impairs his value, incapacitating him from setting back and holding heavy loads coming down hill, and he then commences to break up. At this stage muscular atrophy becomes evident, but it appears to be a generalised condition rather than the local muscular atrophy indicating changes in the spinal cord anterior to their trophic centres.

Case I. Female donkey (aged), in good condition, but badly affected with stringhalt both hind. When first taken out of the stable the hocks were jerked up at every step, but after a few moments exercise the movements became less marked.

Tests Employed. Blindfolding neither increased the movements nor caused stumbling. Eye reflexes apparently normal, sensation as demonstrated by pin pricks undiminished, myotatic irritability normal. Action of the bowels and bladder normal (thus showing that there was no interference with the vital reflexes in the lumbar region of the cord). Stroking of the panniculus caused twitchings.

Post-mortem Examination. Naked eye. All the viscera appeared normal; the brain was exposed and removed, both it and the meninges appeared healthy; the pia arachnoid was delicate and transparent all over, and the blood vessels and sinuses apparently normal. The spinal cord was exposed in its entirety and removed, both it and its meninges showed no change. The median and sciatic nerves were excised, and to the naked eye were normal. The tendinous insertions of the peroneus, flexor metatarsi, and gastrocnemius muscles were also incised and removed for microscopic examination: to the naked eye they looked healthy. The various joints of the hind limbs were also healthy.

Microscopic Examinatiou of the brain, spinal cord, nerves and muscles, failed to reveal any pathological changes.

The following methods were employed :—

The nervous elements were stained (1) with Haemalum and Van Gieson to show whether sclerosis was present; (2) by the Weigert Pal method to show late degeneration; and (3) by Marchi to show recent degeneration.

The muscles were stained with Haemalum and Van Gieson, but showed nothing abnormal.

AUSTRALIAN STRINGHALT.

This disease, which in some respects resembles stringhalt in Great Britain, appears to be peculiar to Australia, where it affects large numbers of horses in certain districts. We are indebted to Professor W. T. Kendall, of the Melbourne Veterinary College, and Mr. Edward Stanley, F.R.C.V.S., for particulars of this malady. The following extracts and notes are made from veterinary reports furnished by them to the Minister of Agriculture for Victoria.

In 1865 or 1867 the disease appeared in Victoria, it seems to follow in the wake of Agriculture, and breaks out most frequently in paddocks which have been ploughed and then laid down in grass. As a rule it prevails in low lying rich lands, and it always comes on suddenly without any assignable cause, and usually about the autumn.

Professor Kendall considers that the malady has three forms, namely, local, general, and acute general.

The Local form is the most common and "is characterised by a peculiar jerking action in one or both hind limbs, which may be so slight as to be only noticeable when the animal is suddenly turned round or made to go backwards; but in more severe cases, both hind legs may be so badly affected that progression can only be accomplished by a succession of bounds and plunges extremely painful to witness." In cases where both legs are badly affected, when the horse desires to move forwards, the hind quarters are suddenly elevated, and one hind leg is violently jerked upwards, sometimes so high that the foot strikes the belly and remains so drawn up; the other is then brought up in a similar manner, the former being at the same time suddenly brought to the ground with great force. At other times both hind legs will be jerked up simultaneously, or in quick succession, and the hocks remain flexed to their utmost extent until the haunches almost reach the ground, before the horse has power to bring the feet down.

The appetite is almost invariably good, and the digestive and urinary organs appear to perform their functions properly. This form of the disease seldom proves fatal, and affected animals gradually recover without treatment in from 12 to 18 months.

General Form. The fore limbs are generally affected as well as the hind ones, but instead of being jerked up like the latter they are carried stiffly forward, the knee being scarcely bent at all. When undisturbed, a horse affected in this manner may graze and get about without much difficulty, but cannot get along at all when chased or excited, but begins to plunge and flounder about: some fall, and others after making violent struggles to get away, come to a sudden standstill, their heads being elevated and nostrils dilated. If near enough the heart may be heard beating violently.

Acute Form. The symptoms differ considerably from those of either of the other forms, but as all three are found affecting different animals in the same mobs at the same time and under precisely similar circumstances, it is evident that they are only modifications of the same disease.

There is a dragging in all the limbs and frequent stumbling, the hind fetlocks knuckle over, and the thighs have a thin wasted appearance when viewed from behind. If the horse is made to move any distance he breaks out into a perspiration. The attack is usually very sudden; should the horse fall or lie down he may be unable to rise, and struggles violently, but if raised by means of slings he can both stand and walk, and if properly attended to he usually recovers.

Treatment. Change of pasture, as well as of locality is one of the surest means not only of preventing the disease, but also for hastening recovery in those already affected. Medical treatment is of doubtful benefit.

Causes. Eating of so-called dandelion, hypochoeris radicata, metallic poisoning from venadium, intestinal worms, rheumatism, ticks and flies, have all been credited with being responsible for the disease, but upon close investigation the evidence necessary to incriminate any one of these has been found wanting.

SHIVERING.

Shivering may be defined as an equine disease manifested by irregular and involuntary movements, generally of the hind quarters, accompanied by *tonic spasms* of certain groups of muscles, with marked difficulty or even inability to perform certain movements.

Symptoms. In the early stages the disease is extremely difficult to detect. Beyond a suspicion of immobility and want of control over the muscles of the hind quarters when the animal is called upon to back, little else can be observed. Even these symptoms are frequently intermittent and cannot be produced by the most severe tests. An average case of shivering may show no abnormality while the horse is walking quietly forward, but when excited, or suddenly called upon to stop, or set back, or to start forward when yoked, powerful tonic contraction of the gluteal and thigh muscles occurs, the horse arches his back and appears to grasp the ground with his hind limbs as if he were in danger of losing his equilibrium. If the hand be placed upon the gluteal and adductor muscles of the thigh while the horse is forcibly backed these will be felt to become rigid, and well marked tremors can be seen as well as felt. The tail is frequently elevated and works somewhat in the manner of a pump handle, although the degree of elevation varies considerably in different cases. If turned quickly in a short circle the limbs move in a stiff inco-ordinate way, and in some cases excessive abduction and flexion of the thighs occurs, and the pelvis sways in a peculiar manner. Any attempt to pick up the hind limbs meets with considerable opposition : if successful in the attempt the muscular tremors can be felt varying in intensity. This feature is frequently the premonitory symptom, hence the shoeing smith is commonly the first individual to recognise the existence of the malady.

Many shiverers show decided symptoms when taken to the watering-trough, or better still, when offered a pail of water on the ground. They become excited, and upon extending the neck muscular spasms seize the hind quarters, the fore feet remain implanted on the ground, the body sways backwards, the back is arched, and the tail jerked upwards. This behaviour, although typical of the disease, is not constant, as there are many bad shiverers that face the watering trough without a tremor.

Acute Shivering. This form is fortunately somewhat rare, still occasionally such cases are encountered, and may terminate fatally in a few hours. I have in my mind at the moment an interesting case. A contractor purchased a lorry horse at a sale, warranted a good worker, but on yoking him was suspicious that he was a shiverer. The horse was sent a journey of 9 miles with a light load, and on the return journey had to bring back a heavy load of sand. The weather was hot and the road hilly; before he had gone far on the return journey the muscular spasms became greatly aggravated, he got highly excited, and broke out into profuse perspiration. Notwithstanding these symptoms, the driver continued on the journey until the horse came to a complete standstill, and had to be unyoked. His distress and excitement were painful to witness: he showed spasms of the muscles of the hind quarters, fore limbs and neck, while the lips, ears and eyelids twitched convulsively, and he swayed on his legs. He was returned to the seller, but died the same night.

Debilitating diseases, more especially influenza, frequently cause marked aggravation of the disease, the animal in some cases being hardly able to come out of the stable, but as convalescence proceeds these symptoms usually disappear. A sudden fright or shock may also produce marked symptoms of shivering in a horse which hitherto has appeared free from the affection: thus a railway journey has been known to develop latent cases of the disease, and I have known street accidents to have a similar effect. These facts are quite in keeping with like experiences in the human subject, as for example terror, shock or grief precipitating latent insanity, chorea or other nervous affections. It is, however, recognised that such causes are not sufficient in themselves to produce the disease without a previous susceptibility, which is often markedly hereditary. Docking the tail has been credited with similar effects, probably the nervous shock and subsequent hæmorrhage being here the exciting cause. Cases of shivering are occasionally encountered where the fore-limbs are also involved, the knee is not freely flexed, and on attempting to pick up the fore-leg the horse extends it in front of him like a prop, resisting any attempt at knee flexion. When we do succeed in bending the limb, distinct tremors can be felt which exactly resemble the spasms in the hind limb of a typical shiverer. In severe cases of shivering we may meet with muscular spasms of the neck, lips and eyelids, which can be observed when the horse is forcibly backed on a slippery surface, or otherwise excited. The nervous temperament of shiverers frequently shows a peculiar irritability and want of uniformity, one day he works quietly, even sluggishly, the next he is quick, nervous and excitable; he may absolutely refuse to do certain work, but on being put to another occupation for a few days, he returns to the former job apparently much benefited by the change. Some of these facts would suggest that in such cases the brain as well as the spinal cord is implicated in the disease. We have an analogy in the human subject where irritability, emotional excitement, and deficiency in will power (*i.e.*, facile) are frequently associated with diseased conditions where if structural changes occur they are strictly localised to the spinal cord. This is not to be wondered at when we reflect how intimate is the relationship existing between the various branches of the nervous system.

Course of the disease. In almost all cases shivering runs a chronic course, is incurable, and gets worse with age, hence it is an extremely grave unsoundness; hard work, deficient feeding, and heavy carting on the slippery streets of hilly towns tend to aggravate it. Although the malady is not infrequent in farm horses, they appear to last much longer.

PREDISPOSITION AND CAUSES.

Hereditary predisposition is undoubtedly a strong factor in the production of this disease. It is met with most commonly in heavy draft horses, but occurs frequently in the lighter breeds. It usually affects horses when they are put to work, but cases have been observed in young stock that have never been handled. It is said to be more prevalent in Scotland than elsewhere, but this is a matter of opinion, certainly it is not uncommon south of the Tweed.

Apart from the hereditary nature of the disease, there is not much actually known as to its cause, and it is remarkable how little has been written on this subject, some of our standard works containing no reference whatever to the disease. The term shivering is much more appropriate than chorea, because the tonic spasms of so-called equine chorea are perfectly distinct from the clonic spasms of chorea minor of man or of the dog. Shivering has been compared to locomotor ataxy of man, but close inspection shows that they are distinct, the definite lesion of sclerosis of the superior column (*i.e.*, sensory) of the spinal cord being absent. Blindfolding does not exaggerate the symptoms nor does it interfere with balancing, neither do the eye reflexes appear to be affected, all of which are typical symptoms of human locomotor ataxy. In the pamphlet on "Diseases of the Nervous System of the Horse" (previously referred to under Stringhalt), Dr. Chalmers Watson reports in detail a case of shivering in the horse in which the stifle joints were both enlarged, and from his description evidently the seat of chronic ulcerative arthritis; he compares the appearances with tabes dorsalis in man, *i.e.*, locomotor ataxy, and concludes that the peculiar joint lesions were neuroptic in origin. It will be interesting to learn from the discussion if veterinarians have observed any connection between stifle joint disease and shivering: personally I regard the conditions as pathologically quite distinct, although they may exist conjointly.

DIAGNOSIS.

There are few duties so unsatisfactory as the examination of suspected cases of shivering, and so long as the disease retains its intermittent character we will have differences of opinion existing, and contradictory evidence in courts of law. We may back the horse, and turn him either in the hand or in harness, pick up the hind limbs and apply the hammer smartly to the shoes, or test him in the stall or at the watering trough with negative results, yet were we to be present when he rises from slumber we might be surprised to notice the rigid position he assumes, the hard tense state of the muscular system, and in some cases the absolute difficulty experienced in standing steadily until the muscular mechanism accomodates itself to the altered position. Some bad shiverers, when they first rise in the morning, present an appearance not unlike the acute stage of tetanus.

Case II. Grey harness gelding, six years old, 14 hands high, badly affected with shivering involving both the fore and hind limbs. Has been in the possession of the present owner for 18 months, during which time he has become gradually worse; although he is still fit for moderate work. He has a dull look, is sulky, and inclined to bite in the stable. He walks and turns in a very stiff, tetanic-like manner, and when forced back the tail is freely elevated and arched over the back; he does not flex either the hocks or knees to any extent, but appears to hold on to the ground, and drags his limbs. Sensation from the loins backwards over the gluteal region defective, but from the stifle down appears increased, and he kicks out on being pricked. Great difficulty is experienced in picking up either the fore or hind limbs, and the peculiar muscular tremors are very evident *when any one of these* are forcibly held, or the shoes tapped with the hammer. He can drink from a pail on the ground with ease, although the tail becomes elevated and spasms of the thigh muscles well marked.

Tests. Blindfolding has no appreciable effect in aggravating the symptoms, nor does it cause any stumbling or interference with balancing. The reflex action of the iris to light appears normal. Myotatic irritability (*i.e.* tendon reflexes) apparently increased. Stroking the panniculus causes exaggerated twitching. The action of the bowels and bladder are normal, but he shows difficulty in assuming the attitude for micturition, and groans during the act.

Post Mortem Examination. All the organs appeared normal with the exception of the kidneys which were somewhat paler than usual, and the heart which was hypertrophied, weighing 9½ lbs., the muscular fibres appeared healthy, but the mitral valves were thickened and somewhat fibroid. All the joints were normal.

The brain and spinal cord were exposed, and there was some congestion of the latter.

Sections of the brain and cord were stained with Haemalum and Van Gieson, also by Weigert Pal, Marchi and Nissl's Methylene Blue method, but no abnormality could be detected. The superior nerve roots were also microscopically examined after staining with Haemalum and Van Gieson and by Nissl's method, but they also presented a normal appearance.

REMARKS.

It will be observed from the foregoing notes on the post mortem examinations of the donkey and harness cob affected with stringhalt and shivering respectively, that the microscopic investigation of the nervous and muscular tissues failed to reveal any definite structural lesions, so far as it was possible to ascertain by the most approved, thorough and recent methods. While these results appear disappointing they undoubtedly tend to disprove that *certain definite structural changes* hitherto recorded can be the essential cause of these diseases. Such absence of recognisable structural lesions, however, is not confined to equine shivering and stringhalt, as many grave nervous diseases in the human family, e.g., chorea, epilepsy and even old-standing cases of insanity, have up to the present time remained unidentified with any definite lesions. Of recent years the theory of the *toxic origin* of many nervous affections has steadily been gaining ground, the symptoms exhibited being attributed to toxins present in the body acting upon the central nervous system, and the liability to such intoxication being distinctly hereditary. Little is known, however, regarding the nature of these toxins or whether they are formed as a result of faulty metabolism, or perverted secretions.

When we review the whole circumstances of Australian stringhalt, as recorded by Kendall, they appear to strongly support the toxic theory.

In conclusion, I desire to express my gratitude to Dr. Ivy McKenzie, M.D., Director, Western Asylums Research Institute, Glasgow, and Dr. Robert Macnab Marshall, M.D., Senior Assistant Physician, Royal Asylum, Gartnavel, Glasgow, who are responsible for the microscopic part of the work, for the enthusiasm with which they interested themselves in the subject, and for the painstaking manner in which they carried out the intricate duties of the research.

LITERATURE.

"The Practice of Veterinary Surgery," Moller and Dollar.
"Law of Horses," Oliphant.
"Veterinary Notes for Horse Owners." Hayes.
"Diseases of the Nervous System of Horses." Chalmers Watson, M.D.

DISCUSSION.

Prof. G. H. WOOLDRIDGE: It is a very difficult task that falls to my lot in opening the discussion on this paper presented by Prof. McCall. In the first place I must confess to some disappointment in the paper, for I had hoped that some light might have been thrown upon the causes of these two very troublesome conditions of which we know so little. We must all admit that they are very difficult problems to tackle, and now that Prof. McCall has commenced work on this subject I trust he will continue it, and at a not very distant date be able to bring before our notice evidence that will throw light on the true nature of stringhalt and shivering, Prof. McCall mentions a very interesting feature in connection with both these conditions, namely, that they become more evident when subjects become infected with some debilitating disease, such as influenza. I may say that my experience supports that view, and some cases that were previously unsuspected become evident in the latter stages of one of these very debilitating diseases.

I think most of us here will agree that the condition is of nervous origin, and that it may be hereditary. It is an undoubted fact that symptoms somewhat resembling stringhalt may arise from other causes. According to the late Prof. Dick, in some cases that came under his observation tumours in the lateral ventricle were found, and he regarded them as being the cause of the symptoms, but as to whether they were actually the cause of stringhalt may be another question. The fact is that the two conditions were concurrent. The late Prof. Williams recorded cases of stringhalt in which, on examination, he found exostosis of the shaft of the ilium involving the sciatic nerve, and he regarded that in those cases as the cause of the trouble. I have myself seen cases—and no doubt most of those present have also seen cases—in which the peculiar catching action of the hind limbs has been observed, and at first appeared to be stringhalt but on closer operation was proved to be due to other things, such as injuries to the foot. I have noticed it especially in some cases of toe sand-cracks in the horse. Cases which at first observation might have been put down as stringhalt were subsequently found to be due to other causes.

I do not think for one moment that spavin is a cause of stringhalt, and in that I agree with Prof. McCall. The two conditions, of course, may be coexistent. With regard to the muscular origin of the affection, I am of the opinion that it is only muscular in origin in so far as the muscles are under the control of the nerves supplying them; that is to say, it appears to me to be nervous in origin.

As to its association with chorea, I prefer not to consider stringhalt as the same condition that we generally appreciate as chorea in the lower animals. In chorea the symptoms appear to be more or less continuous, even while the patient is at rest; in stringhalt the symptoms are only observable when the animal affected is progressing, either forwards or backwards or being turned in a circle. An important symptom omitted from the paper is the force with which the foot in many cases is brought to the ground during progression. Sometimes the force is so very great that in the particular animal affected it is impossible to keep a shoe on, and in other cases the shoe is actually broken in two.

Nothing has been said with regard to treatment, no doubt because any treatment must be empirical in the absence of any knowledge of the cause. Palliative treatment may be of some service. Since a debilitated condition of an animal affected aggravates the symptoms, it might be reasonable to expect that attendance to the general health and condition of the animal, in bad cases at any rate, might have some effect in relieving the intensity of the symptoms. That I think will only be so in exaggerated cases, most certainly not in the ordinary mild cases of stringhalt. Section of the tendon of the peroneus just below the hock on the outer side of the cannon may be practised, but obviously this does not remove the cause. It frequently, however, reduces the severity of the symptoms by diminishing the combined power of the flexors of the hock, and in some cases the improvement is seen immediately. In others it is seen after several weeks, but in others there is no improvement whatever. The only way in which it produces its beneficial effect, I believe, is simply by reducing the muscular power of the limb—not by removing the cause of the condition in any way.

I should like to know something more about the so-called enzootic stringhalt as met with in Australia. When reading accounts of it I have wondered if it was not possibly some condition embolic in origin, possibly verminous. I have not seen it, of course, since it only occurs in Australia. In connection with two of the forms which were described in the course of the paper, the so-called general form and the acute form, I am far from convinced that they are in any way related to stringhalt. They both read very much more like rheumatism than stringhalt, and there was no suggestion that there was any resemblance whatever to stringhalt in the symptoms.

I am afraid that in connection with shivering also we are not any nearer a solution of the problem. The description of the symptoms is very good indeed, but I was struck with the statement that shivering is frequently intermittent. I must say that

I have not experienced intermittent shivering. It may occur, and if so it is a very important matter indeed from the point of view of examinations for soundness, since shivering is obviously an unsoundness, and we may get into trouble by passing a horse at a time when no symptoms of shivering are to be observed but in which symptoms may be found on a subsequent occasion. I should like to hear the views of the more experienced members on that point.

With reference to the case of so-called acute shivering recorded by Prof. McCall, it would have been interesting and perhaps instructive to have heard something of the post mortem appearances in that case—a case in which the symptoms were developed very rapidly, and the horse died in the course of the next night. From the symptoms it appears to me very much more like a case of acute cardiac affection than a case of shivering.

That point leads me to refer to two cases that have recently come under my own observation. In each case the symptoms were exactly the same, and in each case the course of the disease was the same. The symptoms were those of advanced shivering with loss of control of the movements of the hind legs, swaying from side to side, threatening to fall almost at every step. It was absolutely impossible to back the horses, and in fact the condition was just that which has caused French veterinarians to call the affection "immobilité." The symptoms were those of partial paraplegia. On examining further there was marked cardiac disturbance, an impulse against the chest wall varying in intensity in successive beats, the intervals of the beats were irregular, and frequently the second sound of the heart beat was duplicated. No murmurs could be detected. Those cases were each given a purgative and then put on a course of cardiac tonics consisting of digitalis and nux vomica, with the addition of potassium iodide with a view to assisting in the absorption of fluids, if there were any, in the neural canal. Both cases improved so much in three weeks that they were able to go back to work. They were not completely recovered so far as the symptoms of shivering were concerned, but they went back to work. I was rather surprised and pleased in a way, although at the time they first came under my notice I strongly advised slaughter, and said they were not likely to be of any further service. Apparently my prognosis was wrong.

In connection with the causes of shivering Prof. McCall does not mention the idea so commonly held by agriculturists and some veterinary surgeons that it may be caused by an injury to the back when quite young, either at the time of birth or at casting for castration. For my own part I do not believe this to be a common cause of shivering. The condition is met with in mares which are not castrated and so can be absolved. In a particular district in Staffordshire with which I am acquainted, this view was held very largely until the standing method of operation for castration came into vogue, and agriculturists refused to have horses cast at all. Then they expected that shivering, which was common in that neighbourhood, would disappear. But after the operation had been done in that way for several years they discovered that shivering was just as common as ever, and they have eventually discarded the view.

Prof. McCall asks for an expression of opinion as to the connection between stifle joint disease and shivering. My opinion agrees with his; I believe them to have no pathological connection, although of course they may be coexistent. I am afraid Prof. McCall is very vague indeed in his description of the post-mortem appearances of his cases. He says in connection with a shivering case, that the brain and spinal cord were exposed and there was some congestion of the latter. Surely that is not a sufficient description of the condition of the neural canal in a disease that is being particularly investigated for some lesions of the spinal cord. We are not told whether there was any effusion, whether the meninges, or what particular meninges, of the cord were affected, beyond the fact that there was some congestion—a condition that might easily be due to the method of slaughter.

May I again express the hope that Prof. McCall will persist in his endeavours to describe the nature and cause of these conditions, and that his labours will ultimately meet with success.

Mr. C. **Sheather**: I have had very little opportunity of studying this paper, but it has struck me there are a few points which might with advantage be alluded to. On the very first page I have marked the definition of stringhalt as a spasmodic and excessively rapid flexion of one or both hind legs. In my experience of some thirty years I have seen a large number of cases of stringhalt in the forelegs. It is not confined to the hind limbs. It is not an uncommon thing to see a horse going with uneven action in front; horses free from lameness, but at the same time with an elevation of one knee considerably higher than the other. That was very strongly brought to my notice in Yorkshire two years ago, when I was shown a two-year-old absolutely useless from stringhalt in one fore limb. He had been bred out of a stringhalt mare from a stringhalt stallion. He could scarcely bend his knee, or go at all. It is one of the most hereditary diseases that exist; in fact, all nervous diseases are, I think, strongly hereditary; and unless care is taken the disease will increase to a very great extent. In showing horses, if a man is ambitious to get a very fine horse he may think a touch of stringhalt is like a little whisky in the water, adding a little zest to it

—and so it does, but it is very unfair for a horse having stringhalt to gain a prize over one who is absolutely sound in his action. Stringhalt in my experience has been produced in very extraordinary ways. I have seen animals apparently absolutely free from it. They have been blistered on the forelegs and stringhalt has followed, and remained. Or they have had an attack of colic and stringhalt has followed and remained. So-called influenza and other febrile diseases will sometimes produce stringhalt which remains. This rather leads me to think that the origin of stringhalt must not be looked for in the spine alone, but probably there is some central reason for such varying causes leading to the exhibition of this disease.

Shivering has also a fascination for me, and I have seen some extraordinary cases. I can give Prof. Wooldridge a very fine instance of intermittent shivering, a little anecdote that ought to impress itself upon his mind for ever. Fourteen or fifteen years ago I was entering a dealer's yard, George Russell's, the horse dealer, in the Edgware Road, and he said, "You are the very man I want to see, I want you to look at a horse for me. I have a dozen certificates and I want yours as well. I sold this horse three months ago to a firm of brewers for a big sum and he was returned to me as a shiverer. I have any amount of certificates to the effect that this horse is not a shiverer, and I am going to make them take him." He took me to see the horse, and the very first thing the horse did as we entered the stable was to shiver in front of both of us. He had been in that stable for three months, watched by men who knew shivering as well as any skilled man would do, and he had never shown the slightest sign of it. That is a very startling case of intermittence in shivering.

I will give you another illustration. Last year a horse was brought to my stable by a builder to be examined for soundness. I was very careful in going over the mare, and I tried all the usual tests for shivering, tried her in the stall and out of the stall, and the builder said, "What do you think of the new mare?" I said, "I can detect no unsoundness in her." He said, "I shall buy her, but I want you to hear what my man says." He turned to his man and said, "What did you notice this mare did yesterday morning?" The man replied, "When I was putting the bridle on she went like 'that' with one hind leg." I said we would see if she did it then, and we tried her again and again. When the mare had the bridle put on in my stable, and her head was half turned and down, the man pushed her a little back, and she shivered again. There is a case in which I could not detect any unsoundness. Perhaps I ought not to say that; we do not pass animals as sound. I had to say that this animal was a shiverer, and I said it is a disorder which a man might not see again for a fortnight, and that it might be shown to half-a-dozen veterinary surgeons who would come to the conclusion that I first came to. It is one of the most disastrous diseases for a veterinary surgeon to meet, and I could multiply the instances of intermittent shivering to no end.

Mr. J. J. Bell: I am pleased to hear the remarks made by Mr. Sheather, and I can corroborate every word he says. I have been engaged in a very great number of actions at law where sometimes we have had as many as a dozen or sixteen veterinary surgeons, and the differences of opinion given by the profession in connection with the disease are so varied that there is no question it is a difficult matter. With regard to stringhalt, I remember a very important case in an animal we gave 500 gns. for in Ireland. The first time he was used he came over on to his back, hit the back of his head, and from that day he was affected with stringhalt. Otherwise he was a perfectly sound horse, and none of his progeny showed any signs of it. It was a case of accidental injury to the head that produced stringhalt. With regard to shivering, I have known animals up to two or three years old that have never been observed until they have been sold, and then after a railway journey they became shiverers. These cases invariably follow, as Mr. Sheather said, from hereditary causes. We can always trace the history of these cases.

Mr. R. Hughes: I heartily congratulate Prof. McCall on his endeavour to give us some information. Yet I felt somewhat disappointed, for I anticipated that the pathology of the disease would be more elucidated. There is no doubt in our minds, I think, but what these are diseases of the nervous system of the horse. It only requires something unaccustomed—unusual work or a fright or some concurrent disease—to develop probably what is latent in the system. I have known cases where Shire colts have been removed by rail and on arrival at their destination, or within a day or two, have developed shivering, although it had never been noticed before.

Coming from a breeding district, I have numerous instances to show that shivering is the most hereditary unsoundness we have, and I expected that Prof. McCall would have endeavoured to call attention to the close relationship that exists between the different nervous unsoundnesses of the horse. I maintain that if a dam is affected with either shivering or roaring the progeny may develop one or other of the diseases. I have known mares that were shiverers to breed roarers before they were four years old. Likewise I have known mares which were roarers have a percentage of their progeny affected with shivering. Therefore, I should like to ask Prof. McCall whether he has made a study of this point, and whether he has had any experience of the close relationship existing between the different neuroses of the horse.

As to the pathological conditions, I have had a few opportunities of making post-mortems, but I must admit that I have never been able to satisfy myself that I could ascribe this unsoundness to any particular lesion, so that my endeavours in that respect have been quite fruitless.

The important point is the intermittency of shivering in relation to soundness. I have had many experiences of perfectly honest gentlemen passing a horse one day and another day the horse would show signs of shivering, and hence I contend that we should have an open and unbiassed mind upon that point. I do not care whether you take the negative or positive way of expressing an opinion; if I find no unsoundness in the animal I generally say that I find the said animal sound. I am only requested to give an opinion, and whether I say sound in my opinion, or whether I say that I fail to detect any unsoundness, it is one and the same thing, I think. I had three particular instances of this. Some very valuable Shire horses were going from our neighbourhood, and I passed two of them sound 13 weeks before Wrexham sale. They were making between 60 and 80 guineas. I had an opportunity of examining them again in assessing their damages for the vendor, as I was asked to arbitrate between the buyer and the seller. In two instances the horses were slight shiverers. As has been said, it only requires something unusual, fright or a railway journey, or even tramcars, to develop the disease, but to my mind the horse would have the disease in a latent form and this change of circumstances, or an acute disease, produced the shivering already existing.

Mr. J. Cameron: My experience on the subject of this paper is only of the ordinary type. So far as the post mortems are concerned, and so far as the ordinary practitioner is associated therewith, I think discretion is the better part of valour, and I do not find any fault with Prof. McCall because he did not give us a very minute pathological description of the post mortem. I recognise that the Professors of our veterinary colleges in many cases are just as much in a difficulty in getting their daily living as the ordinary practitioner, but there is one conclusion which I think the whole profession should come to on the subject, viz., that to arrive at a satisfactory conclusion and an elucidation of the subject requires some Government assistance. Why does the Government not give a grant to our Professor of Pathology that he might devote his time to the work? It is not only a disgrace to us as a profession but a disgrace to us as a nation that we have no means of going into research in connection with subjects like this. I think this is one of the diseases which we could use as a lever to raise Government assistance (Hear, hear).

Mr. E. W. Hoare: The excellent paper which Prof. McCall has put before us emphasises the fact which was already well known—that we know practically nothing about either shivering or stringhalt in the horse, and this of course would apply to other diseases as well as stringhalt and shivering. There are a few points in connection with stringhalt to which I should like to draw attention, points which might be termed omissions in the paper, so commonplace, perhaps, that Prof. McCall might not have thought it necessary to draw attention to them.

First with regard to wounds. A horse with stringhalt might get a nail in his foot and that nail might set up enormous irritation, far more irritation than in the ordinary horse. So serious have such cases become in some of the stables I attend, that I attribute greater seriousness to wounds in the foot in animals with stringhalt that I do to the disease itself.

I have seen cases of stringhalt in the fore limbs; they are not uncommon and they generally arise from some form of injury. With regard to the damage which stringhalt does to a horse from the point of view of soundness, I do not consider that slight stringhalt is of such great importance, especially in a hunter or a harness horse, because I have known horses carry their owner well for years with slight stringhalt which has never increased.

With regard to shivering, it is a question of importance whether there is not more than one form of shivering. Many veterinary surgeons combine all forms of nervous diseases under the head of shivering. The text books differentiate such terms as immobilité, injury to the spine, and that sort of thing, but I think there is no doubt in the world that for practical purposes they can very well be termed shivering. I am quite sure that most of us have had a bitter experience in discovering that intermittent shivering does exist, and not only that, but that a horse may show no symptoms in the morning and be a shiverer in the evening.

In the post mortem appearances recorded by Prof. McCall I think it is very disappointing that no more definite particulars were obtained than a mere congestion of the spinal cord. With regard to docking, I think there is no doubt whatever that docking will render the symptoms evident. Prof. McCall says that the hæmorrhage after docking has some influence, but I can hardly see how that could be because the hæmorrhage after docking is so trivial in amount. Prof. McCall mentions one case in which a horse went out in the morning with what was supposed to be shivering and developed certain symptoms on the road and died that same night, and he calls that acute shivering. I should be more inclined to say that the horse may have been a shiverer and he had contracted an acute spinal disease afterwards. With regard to the toxic origin, of course toxæmia is a most convenient term to the pathologist. When a man does not know anything of the origin of the disease he calls it toxæmia, ultra-visible organisms. But I do not think that any man could agree that toxins cause either stringhalt or shivering. Another question I would mention more for the sake of getting information, and it is this: Does the fact of a horse being a shiverer render it dangerous to cast it for operations? Many people imagine that it would not be safe to cast a shiverer, but my own experience, from casting a great number of horses that are shiverers, is that they have always got up again.

Mr. C. Sheather: If I might be allowed to say another word I have rather an interesting case to relate in connection with shivering. I have said that I considered in all probability the mischief would be found to be central, on account of the various outside affections resulting in shivering. A case is brought to my mind by what Mr. Hoare has said in connection with casting. We know that thoroughbreds very rarely suffer from shivering—I have seen one or two, but it is extremely rare. With another veterinary surgeon I was asked to go down and give a little assistance to an owner of a mare that had a cankered hind foot. She was a three-year-old thoro'bred mare, and it was necessary to cast her because she was difficult to deal with. The mare was cast, not in the way I should have liked to have seen her cast, and she gave no evidence of injury in the casting. The foot was thoroughly attended to, and she was allowed to rise. She went off the tan bed four steps, and then distinctly shivered; then she walked on perfectly well and went into the box. That night she was destroyed for fractured spine. It is interesting to see that a fracture of the spine caused shivering in an animal which previously had no taint of it. For those who want to study the thing pathologically it might be a fact of some service.

Mr. Ainsworth Wilson: In thanking Prof. McCall for his paper I should like to say that I sympathise with him to a certain extent, because he is dealing with a subject about which we know very little indeed. He is doing pioneer work in connection with it, and I hope he will continue his investigations. With regard to the definition of stringhalt already referred to by Mr. Sheather, the disease does occur in the fore limb, but I think we are all agreed it is certainly rare. My experience is quite limited, but I have seen altogether only three cases in the course of my life in the fore limb, and in one case the horse passes my door at the present time every day. In his case the off fore leg was affected, the knee was extremely flexed, and the foot lifted very high from the ground. The symptoms in each case were very pronounced.

One point has not been referred to in connection with the intermittency of shivering and that is that in all animals—certainly in two-year-old Shire colts—you will sometimes notice that they shiver when they go to water and at no other time. I have two cases in my mind in which two-year-old Shire colts showed very slight shivering in my presence. No other symptoms were noticed, although I saw these horses working every day, until the animals were between four and five years old and were put to very hard work, and then they developed ordinary symptoms of shivering, which remained permanent. I took the opportunity of examining these animals several times and could never make them show any sign of it. Reference has been made to the influence of railway travelling and febrile diseases and other disturbances of the nervous system which bring out symptoms of the disease. All of us, I feel sure, have been cheated by shivering. I examined a horse not very long ago, and could detect no sign of any disease whatever, but to my surprise and disgust the animal was returned, and I had to go down and examine him on the owner's behalf, and give a certificate that the horse was a shiverer. He had been travelled by road 16 miles to auction and from the auction he went to London by rail. He was sent from London back to the auctioneer, and symptoms of shivering were very apparent. I agree with previous speakers that it is a matter for regret that Prof. McCall has not been able to discover very much with regard to these nervous diseases, but really I do not think we anticipated he would discover a great deal. It is a very difficult task he has set himself, and I wish him every success; I hope he will go on and prosper.

Col. Steel: Prof. McCall has not mentioned any particular age at which horses are most subject to shivering and stringhalt. I have a strong idea that three years old is the age at which they are most troubled, and I think the irritation produced by the great changes that are undergone in the mouth at that time is very frequently the exciting cause of many, if not all, of these nervous diseases. I would include under these nervous diseases a very patent fact to those who have had much acquaintance with roarers—that roaring comes on at that particular period.

Mr. Hall: I should like to record a case of stringhalt I saw only last week. The animal had a mild attack of lymphangitis in the stringhalt limb, and the mischief was exaggerated so greatly that it necessitated slinging the animal. Such a case has not happened in my experience before, and I thought it would be of some interest to mention it.

Major General SMITH, C.B. : I am sorry to say that I know nothing about either of these diseases, but the Chairman invited me a short time ago to make a few remarks on the subject, and I felt it might be well to lay before you one or two points that have occurred to me during a somewhat long experience, even should they be of a negative character. With regard to stringhalt I may say that we get a small number of cases in the service, and although some of them are acute, my experience agrees with that of a previous speaker who said that the disease did not very seriously interfere with an animal's usefulness. I have, however, in a few cases known horses suffer to such an extent and the fetlocks flexed with such violence that the hair was removed from the part.

There is one point which I think has not been referred to by previous speakers, and that is an early indication that may be found in cases of stringhalt. A horse may show no sign whatever of the affection when you are examining him for soundness, either in travelling from you or towards you, but he may show it very distinctly on the turn. A horse that shows no indication whatever of stringhalt when examined on ordinary ground will frequently indicate the disease when he is trotted on stones. I do not know whether this agrees with the experience of the members present, but it is a point on which we might hear something further.

Some years ago I was asked to try the operation of peroneal tenotomy for the relief of stringhalt, but although it was done in several cases, there was not the slightest indication that it was of any value.

It is very curious that although a considerable number of horses pass through our hands every year I do not think we ever meet with a case of shivering. Of course we sometimes meet with them before they come into the service, and they are naturally rejected unless we get "let in," and there is every reason why we should occasionally overlook it judging from what we have heard this morning. It has been stated that many cases of shivering may result from previous injury. For instance, there is the case of Mr. Sheather's where shivering symptoms were shown when the horse rose with a fractured spine. I need hardly remind you that a very large number of cases we have to treat in the service consist of injury, and I have never seen a case of shivering result from this cause. I think it is very important that evidence of this sort on the other side should be brought forward, because some of the facts we have heard this morning are very striking indeed. I should like to congratulate Prof. McCall on his endeavours to throw light on an extremely obscure subject. He should receive our sympathy rather than adverse criticism, because there can be no doubt whatever that investigation into the diseases of the nervous system is the most difficult line of pathological enquiry that can be adopted.

Mr. H. Sumner: I hope as a result of our discussion we have proved a case for the National Society to endeavour to do something to encourage workers to elucidate some of the problems of shivering and stringhalt in horses. Our thanks are distinctly due to Prof. McCall for coming forward and showing us that he is deeply interested, and possibly will be prepared to go on upon certain conditions. When we come to look at the conditions necessary we shall be probably appalled at the expense. We need physiologists, and neurologists, and patients, and all are expensive matters. But I do think that an attempt might be made to acquire typical stringhalters, and put them under a definite scientific investigation, both ante and post-mortem. As a town practitioner associated with heavy horses it is naturally my lot to attend to many of these cases. Much has been said as to what might be called shivering, and allusions made to injuries to the foot, railway journeys, unusual circumstances, but as more or less scientific men I think we should look upon these as being the exciting causes developing the latent neurosis. Our country practitioners get the credit of cutting their colts too short, or cutting their colts too long, and having shivering as a result, and surely at what is possibly the first crisis in that young animal's life he may be particularly susceptible to the development of this neurotic affection. Of course the intermittent shivering has done us all in, but I cannot think of a horse that was intermittent in his stringhalt. "Once a stringhalter always a stringhalter." What I would like to know is whether it is a common opinion that shivering may be intermittent, but is always present after it has once become developed. Perhaps you gentlemen may know more from a wider experience than mine, but I do believe that these neurotic horses can be put under some particular treatment which will enable them to undergo a more or less severe veterinary examination at a sale yard or an auction ring.

There are several collateral symptoms which have always interested me, one of which I was put upon by a thoughtful layman, namely, the consideration of the mobility of the tail in respect to the movement of the hind limb. I do not mean cocking up the tail in a well marked shiverer, but where you are examining a horse in a very particular examination—it may be a horse or mare with the idea of stud purposes—it does make one particularly anxious to watch all movements, and I believe that where we see the movement of the tail particularly responsive to the movement of the hind limbs, we may, as the Scotchman says, "hae our doots." A case in point deceived me upon a very recent occasion. I was called up to a sale yard hurriedly to examine a particular mare. The period of time when they could be exercised and tested had passed, and I had simply the opportunity of making the examination in the stall, turning her over from side to side, handling her joints, striking her, and such like. In doing so I noticed that the mare was rather drowsy in appearance and I was told it resulted from a long journey she had had that morning. She looked to me, however, as if she had been "nobbled." I said to the man who was there, "Just stand on one side—I should like to take her temperature." I took the temperature with the thermometer in the rectum, and my hand upon the tail, and I got a curious little twist of one of the coccygeal muscles. I turned her and backed her and I picked up the limbs and flexed them—a point on which I place a considerable amount of importance. The mare went into ring and came out of the ring, and when she came out she pulled her leg violently. I do not mean to say that the condition of the tail is a serious one, but I do think that this sympathy in the movement of the tail in relation to the hind limb is one well worthy of our further consideration.

Some of the speakers have gone so far as to criticise some of the points in the paper, especially that wilh regard to post-mortem. I myself do not want to go so far as to criticise in any shape or form some of the cases which our friend Prof. Wooldridge has produced, but I should think it was worth our while to consider whether the cases he has given were those of immobilité proper, want of co-ordination, want of power, or whether they were cases such as we have been considering to-day in which there is an intensity of power.

Mr. A. Spreull: I agree generally with the remarks that have fallen from the majority of the speakers, and consequently did not think it was worth my taking an active part in the discussion, but there is a case which has recently occurred that might be worth telling. It is the case of a yearling that had never been worked. He showed previous to castration, which was done as a standing operation, considerable well-marked symptoms of shivering. The operation made no difference: he still continued a shiverer. What is more he developed a schirrous cord that had to be operated upon. That could not be done standing and he was consequently cast. After the operation he healed very nicely but he still remained a shiverer. Now unless there was something latent, how could that be accounted for in a young animal like that? I think there is no doubt whatever that it results from a latent condition in some part of the nervous system, and in regard to that I had the pleasure recently of listening to a description by a member of the profession who stated with all authority that the diseased condition was due to hyperplegia of the lateral ventricle. What he meant to convey by that I do not know.

I am quite at one with regard to the intermittency. I have known cases of stringhalt and shivering that have been intermittent. You will get a horse that you know to be a shiverer or affected with stringhalt, and at one examination you find he shows it and at another examination, perhaps on the same day, you will find he shows no symptoms whatever. With regard to the causes of exaggeration, cases that are travelled by rail have marked increase in the symptoms when they have just arrived. An injury to the foot is a common and fruitful source of development of shivering when you least expect it. I have seen cases of suppurating feet cause development of the symptoms of shivering which previously to that was not noticed. The same results occur in connection with influenza and any nervous or debilitating disease.

Mr. J. B. Dunlop: I have to congratulate Prof. McCall on his paper, because I think he has described the symptoms of stringhalt very well. Of course, we all know the symptoms of stringhalt in its most aggravated form, but what veterinary surgeons want to know is how to recognise the symptoms in the very early stages. In examining horses for stringhalt I have always paid particular attention to backing them rather quickly and then pulling them forward rather suddenly and observing the first step. In that first step I have often recognised stringhalt when I could not perceive any other symptom. There was no doubt of these cases being stringhalt, because the symptoms became very apparent afterwards. I have also always paid particular attention to the elevation of the tail, especially in backing, in examining horses for spinal diseases. I believe that these cases are neurotic in their origin, and what we describe as causes are simply exciting causes in the development of the disease. I could not quite agree with the speaker who said that toxins would not cause certain nervous diseases. I believe there is scarcely a disease but is aggravated by toxins. It is very difficult to prove it. I have observed cases of influenza where horses are fed on hay that had been heated by exposure to the weather, and no doubt as the result of eating that hay certain poisons were introduced into the blood, and those cases have been very fatal ; whereas horses that were well fed resisted the disease better. It is twenty years now since I retired from the profession, but I have always taken an interest in it, and not more than a month ago I saw a very marked case of lameness that I attributed to toxins. This horse had the habit of eating droppings, an unusual thing, because horses are usually very clean. We removed the cause, and immediately the effects commenced to cease. I could give many instances of toxins causing lameness, even splints, but that is a very wide subject, and rather getting away from the paper before us.

Mr. J. H. Carter : I should like to express my thanks to Prof. McCall for the admirable paper he has given us to-day. I do not say that there has been a very great advancement in the theory of these two diseases, but I hope it will be the thin end of the wedge, and that in a very short time we may get some Government help to assist us in elucidating the diseases better than we have been able to do up to the present. I do not rise with the idea of throwing any light upon these two diseases, but simply to quote two cases that came under my observation in the last year or two.

One is the case of a carriage horse in which I detected nothing whatever until I lifted the off hind leg to examine the feet, when I felt a quiver. I kept my eyes open and went round the other legs, and again came to the off hind leg and flexed it, but got no result. I had the animal put into harness, and exercised him and brought him back again and put him into the stall. I lifted all the four legs again, and still got no result. I did not know really what to do, and I passed him. The gentleman who took the horse had it about four years, and probably about a month after purchase, when I was calling, the coachman said the horse gave a bit of a click with one hind leg, but that he only noticed it occasionally once or twice. That horse was in the stable three or four years and did good work and never got any worse.

The other case was a cart horse that I purchased for a brewery. In examining the horse I noticed a click occasionally in the hind leg. I remarked it to the dealer who put him in at £15 less than the market value, and I purchased him. I quote this case to show you that we may reject some cases when the probability is that the horse may do good, useful work and never be any worse, because this cart horse is working at the same brewery to day, three or four years after purchasing him, and there has been no advance in the disease. He still shows occasionally just the click in the hind leg. In many cases I think we reject horses that might prove useful, though I do not say that we are in any way to blame for rejecting them. It is no doubt our duty, strictly speaking, to do so.

Mr. S. Villar: I think the writer of the paper and the opener of the discussion are to be congratulated on the success of their efforts, but there is another body which I think might be congratulated, and that is the Provisional Committee who chose these subjects for our discussion—if they had done nothing else that would be worthy of our thanks—because I think I am right in saying that we have had the unanimous opinion of this meeting that both shivering and stringhalt are diseases which are intermittent in character. If it should go forth from this meeting that they are intermittent diseases it would be a good thing for the veterinary profession. It would account for some of those cases in which it is pointed out, more particularly in the case of veterinary surgeons giving evidence, that "doctors differ." We see from this discussion that veterinary surgeons are quite justified in differing, because we have it stated that a gentleman may examine a horse and find him affected with stringhalt and on examining him later on in the day may find him free, and may find him free on examining him in a week's time. Two or more veterinary surgeons may most carefully examine an individual horse and arrive at different opinions. It is a good thing we are able to lay it down that a man cannot be accused of making an absolute mistake because he has described a horse as a shiverer or affected with stringhalt when other veterinary surgeons who have examined him have not found him affected with either of those diseases.

Mr. W. Shipley: I must also congratulate the writer of this paper, because to us in the Eastern Counties it is especially interesting, owing to the fact that we have a great many horses to examine on the farms before they have had the excitement of railway journeys and streets, and it very often occurs that we may miss a case of stringhalt in horses. I think there can be no doubt that it is intermittent, and I would like to relate one case which occurs to me.

Several years ago I was examining at the County Show, with Mr. Bower, of East Rudham, and saw a stallion walk into the ring with very bad stringhalt. I went into the ring to have a look at him, and I called Mr. Bower to see him. He saw he had stringhalt, and sent him out. As a matter of fact the horse belonged to the late King and had always been under Mr. Bower's care. I was speaking to Mr. Bower about it about a fortnight ago, and he told me he had never been seen to do it again although he did it then in a very pronounced manner.

Another case occurred within the last month or two. I was examining in the Show yard when a very valuable horse came in and showed signs of suffering from stringhalt. Another veterinary surgeon was acting and I asked him to confirm my opinion.

Before he got there he said, "I know what is the matter with him—I have seen him do it." We turned the animal out of the yard, and the owner was very much annoyed. The animal had won at least 250 prizes and had never been rejected before. One of the things that amused me very much was that the owner got someone else to examine him, and the certificate stated that he could emphatically say that the disease did not exist. I think that is one of the most ridiculous things for a veterinary surgeon to say about stringhalt. Anyone can say if they see it that it does exist, but that it does not exist it is absolutely impossible to say. I think there is very little doubt about the stringhalt being hereditary. I remember in one case a stallion in our district suffering from stringhalt, and practically every one of his colts were affected, and it took years practically to clear the disease out of the place. That reminds me of some remarks that have been made about colts suffering from stringhalt after castration. My experience is that we get just as many mares suffering from stringhalt and shivering as do geldings, and I cannot see that the operation has anything to do with it, although, of course, I will admit that any injury or surgical operation is likely to bring out the disease when it is latent.

The PRESIDENT: Before calling upon Prof. McCall to reply to the remarks made by the various speakers, I should like to give you my impression of his paper, and also speak with regard to certain points in connection with the speeches. I understand that this essay by Prof. McCall is only the first stage of an investigation into the pathology of the two affections, stringhalt and shivering, and although his paper does not bear any evidence of an extraordinary effort in that direction, I think it is deserving of considerable encouragement on our part. The time at his disposal probably was not quite sufficient, and subjects for investigation are not to be found every day in any one district, and therefore there is a good deal to be said as to the meagreness of the information relating to the etiology of these diseases. I think, however, the paper is a distinct advance on the first stage, and therefore I wish to congratulate him upon what he has done.

But I should like to warn him, and any other individual who cares to investigate nervous diseases, that it would be well for the investigator, particularly if he is not a veterinary surgeon, to have some very intimate knowledge of the appearances of the normal spinal cord, and, of course, the brain as well. When one sends a portion of the spinal cord or the whole cerebral spinal axis to a human pathologist who has no previous experience of the normal appearance of the spinal cord or the neural canal, the information that may be forthcoming after an examination may not be of very great value. Except by very intricate staining of sections it is not possible to obtain much information as to the actual histological change, but there are certain naked eye or microscopic appearances which I think the veterinary surgeon is apt to overlook.

In cases of shivering examined a great many years ago, it was discovered there was a distinct hardening, or as it was termed at the time, a distinct increase of the neuroglia or connective tissue of the spinal cord. However, it is a long story, and I do not propose to pursue it at this moment. I should like to say, however, that I believe an investigation will eventually prove that both stringhalt and shivering have a cerebral spinal origin, and I am inclined to think that the beginning of these affections can be traced in most instances to infection contracted during an attack of strangles or some other disease of early life; and according to the nervous lesion produced by intoxication we have the symptoms sooner or later developed.

Stringhalt, as we commonly see it, is very much exaggerated in importance: far too much attention is paid to the effect or importance of intermittent stringhalt. There are cases that have been alluded to to-day where the stringhalt is distinctly well marked, and there is no doubt that those cases ought to be rejected on every occasion. With regard to the symptoms of stringhalt, it is remarkable that they frequently follow some irritation or wound, even the application of a blister to a leg. I believe there is no satisfactory remedy for stringhalt, surgical or otherwise, and I quite agree with Prof Wooldridge that the operation of aponeurotomy is largely done for the purpose of diminishing the muscular power.

Shivering to my mind is a far more important disease than stringhalt, and it is unnecessary to add a single word as to the hereditary character of the affection. I believe there is what is called latent shivering. Also I believe that shivering is sometimes intermittent, that is to say the symptoms are not continually shown. With regard to latent cases, every veterinary surgeon must have passed numerous horses in his time that were distinctly unsound, if he only knew it, from this cause Intermittent shivering is so common that no veterinary surgeon is to be held liable for overlooking its existence when the symptoms are not plainly to be seen. With regard to the manner in which the symptoms may be developed, if a horse affected with latent shivering suffers an attack of colic you will see the symptoms sometimes develop during the attack. If an animal has a wound, a gathered nail, or a punctured foot, or any other injury to the limbs, the symptoms are very likely to develop. Some people have said the disease is more common in cart horses, and I quite agree with that, but there is no doubt it occurs also in harness horses and sometimes in horses that are three parts bred. If such horses happen to contract a catarrh, an influenza, or any other morbid constitutional disease, the symptoms of shivering are developed and may continue throughout the attack. Then the symptoms gradually subside, and the horse apparently is free from any signs of shivering. With regard to examining a horse and giving a certificate, the usual tests are referred to by the essayist, but I think there is another test that ought not to be forgotten on every occasion, and that is active exercise in the way of galloping. I will give you an instance as an illustration.

I once went to the country where a horse was said to be a shiverer. He had been sold at a public sale and rejected by one veterinary surgeon and passed by another. I arrived on the scene about two o'clock and found the horse in the stable, and submitted him to the usual tests of moving from side to side, backing, drinking from water placed on the ground, walking out backwards, turning, and so on, without seeing any sign of shivering. Then having waited on the premises for three hours, and having allowed the horse an interval of twenty minutes between every inspection, without discovering any symptoms of shivering, I thought the next best plan was to exercise the horse. He was therefore galloped round an adjoining field for about twenty minutes, and when he was brought into the stable and allowed to cool down there was no difficulty in discovering the symptoms of shivering, although they were by no means very well marked.

Another point bearing upon the detection I ought to refer to, because I think one or two speakers rather doubted whether it was quite correct, and that is, whether anything can be done to a shiverer that will diminish the symptoms or cause them to disappear. I am firmly convinced from experience that it is possible to conceal the symptoms of average shivering ; I do not mean a case where the horse is shivering on both hind legs, but where a horse shows slight shivering, and even in confirmed shivering I think the treatment is beneficial. All that is necessary is to place the horse in a loose box or a paddock for three weeks or a month and feed him well, and at the end of that time he will very likely pass a veterinary examination without showing any symptoms of shivering. I believe that is the practice adopted by a certain class of dealer who has to dispose of these animals, and I believe it is very successful. It is during this period of quiescence that the horse is usually submitted for examination, and he passes. I look upon shivering as a progressive disease where the symptoms are acute. It is an exceedingly slow disease if the symptoms are still intermittent or the condition may be regarded as latent.

I do not believe that the disease is produced, as some have suggested, by railway journeys or accidents. What these things do is to excite the animal. The effect of a railway journey or a fall or struggle is sufficient to aggravate the symptoms and bring them into relief. But the horse that is discovered to be a shiverer at the end of a railway journey, and is alleged never to have shivered before—well, I do not believe the story. I believe the horse has been a shiverer but the symptoms have been more or less latent.

Mr. R. Hughes: I think you said that stringhalt was due to infection at some period of life: would you in that statement exclude the possibility of heredity as a factor?

The President: No.

Mr. J. J. Bell: If the President was alluding to my remarks in connection with shivering from a railway journey, I think he has rather misunderstood me. The animal in my mind was the subject of hereditary shivering and the railway journey aggravated it. The case was tried before the High Court of Edinburgh, in the process of which Lord Mackenzie paid me a very high compliment.

Reply.

Prof. McCall: I should like to return my thanks to the various gentlemen who have taken part in the discussion for the very courteous manner in which they have dealt with my paper, with its many imperfections. I came prepared to hear criticisms of a much more severe type than I have received. I note that the treatment of stringhalt was mentioned by Prof. Wooldridge, who said that palliative treatment would be beneficial. I presume he referred to the cases of stringhalt which became manifest in conjunction with an attack of debilitating disease such as influenza, and there, of course, the treatment would not be so much treatment of stringhalt as treatment of the general health of the animal, the raising of the tone of the general system, which has undoubtedly a marked effect on the condition of an animal. The nervous system and the general system run very much hand in hand. I referred in my paper to Australian stringhalt and quoted the three forms, but Prof. Wooldridge questions whether those forms are really the same disease. We find from the Report by Prof. Kendall that these different forms occur in the same herds at the same time and under the same conditions, and therefore I think from that we are entitled to assume that they are modifications of one and the same disease. I reported in my paper a case of what I called acute shivering, but I refrained from giving any result of the post mortem examination because I found no affection of the heart; so that this case was not a similar one to that referred to by Prof. Wooldridge. I did not make any examination of the central nervous system. I think when we get cases of well-marked shivering, spasms of certain muscles, under the influence of over exertion, we sometimes find the symptoms becoming aggravated and the contraction of the muscles becomes marked over the whole body, and the assumption that this is an acute form of the same disease is not at all unwarranted, and certainly has not been at all disproved. Several gentlemen have criticised the brevity of the report of the post mortem examination and I admit it is extremely brief, but as we found practically nothing I did not think there was any reason why I should have wearied the members with details to very little benefit. I have never seen a case of what I would call stringhalt in the forelimbs. I have seen slackness that showed a want of uniformity in the knee flexion, animals who went perhaps an inch or two inches higher on the near fore than they went on the off fore, and had consequently to be differently shod for show purposes, but that action was different in my opinion from the contraction of stringhalt in the hind limbs. Mr. Hoare referred to a prick as being a cause of stringhalt. I think we are all pretty well agreed that although a prick may accentuate or make manifest the existence of this nervous lesion, the injury in itself is certainly not sufficient to account for the cause; it simply magnifies the complaint or makes it more manifest. With regard to the casting of animals being a cause of shivering, there can be little doubt that if we have a hidden nervous lesion the excitement of the casting and the results may be sufficient to make the condition manifest. If Mr. Hoare refers to my paper he will find that I did not say the hæmorrhage from docking was sufficient to account for the lesions—although we may get considerable hæmorrhage sometimes—but that the shock and hæmorrhage might be sufficient. Col. Steel referred to the age at which the lesions of stringhalt and shivering become manifest. In my paper I stated that the age at which these nervous affections most commonly become evident is when the animal is put to work, and that varies in different districts from three to five years. As a rule they become worse with work, especially hard work.

Mr. Sumner raised a very interesting point in connection with a grant for carrying on research into diseases of the nervous system, and I should just like to say that such diseases are extremely intricate. We find that of all classes of disease the least is known about the lesions of the central nervous system. There are hundreds of men dealing daily with affections—insanity, epilepsy and other diseases, and yet up to the present time they have found practically nothing. Most up-to-date laboratories, and equipments of the very best, and yet with their present methods they are able to discover nothing. We should naturally think that in connection with an animal affected with severe shivering or stringhalt we only had to make a few sections of the spinal cord in the lumbar region and we should find sclerosis or some other condition ; yet in two cases to which I referred sections were cut and the most approved methods adopted and the results were absolutely negative. I have to-day received an interesting letter from Mr. Wolstenholme, of Manchester, who writes that he lately had a case of a bad shiverer sent to him by Mr. Faulkner of Manchester. The animal was destroyed and the brain and the spinal cord forwarded to the Medical Officer of the County Asylum of Manchester, who made a very careful examination of all the tissues, and reported he found nothing abnormal. Therefore we have two really bad cases in which nothing at all was found.

But the main object of my paper was really to introduce for discussion the clinical aspect of the disease. Reading over the evidence of court cases it appears to me that it is a great pity veterinary surgeons should come into the witness box, one on the one side and one on the other, and give distinctly opposite evidence. The result is that the magistrates form an altogether wrong idea of the condition of affairs. If at this meeting we have agreed on tbe intermittent nature of these two diseases, as I think we have, with the exception of my friend Prof. Wooldridge, who was somewhat sceptical on the point, we have at least gained one important point. I should have had another post-mortem to report upon but for the fact that animals nowadays are not so easily procured. Up to a year ago it was quite easy to go down to markets in Glasgow and pick up a bad shiverer or one affected with stringhalt for a sovereign, but now the animals affected with these diseases are kept in a stable and fattened and sold to go abroad, and it is nearly impossible to get a horse under £8 to £10.

[The meeting then adjourned for luncheon.]

ACUTE GRANULAR VAGINITIS IN CATTLE, AND ITS RELATION TO STERILITY AND ABORTION.

By Ainsworth Wilson, F.R.C.V.S.

This disease, peculiar to the ox, is essentially a catarrhal inflammation affecting the vulvo-vaginal mucous membrane in females, and the prepuce and glans penis in males.

The pathognomomic symptom is the presence of small nodes or granules; these are found in heifers and cows during the whole course of the disease; they have also been observed occasionally in bulls, while vesicular and ulcerous lesions are entirely absent.

The causal agent is said to be a streptococcus, discovered by Ostertag, found in the deeper layers of the vaginal mucosa, and in the muco-purulent discharge. The irritation excited by the specific streptococcus is followed by swelling of the lymph follicles; these appear prominent on the surface of the mucous membrane in the form of numerous small, firm granules. The catarrh may remain localised in the vestibule and vagina, on the other hand, it may extend forward to the uterus. Untoward sequelæ are endometritis, pyometra, cystic degeneration of one or both ovaries, and hypertrophied corpora lutea. Abortion and sterility are therefore said to be frequently associated with virulent outbreaks of the disease.

In these days of the ubiquitous motor-car, the dairying industry invites the earnest attention of the veterinary profession. It is to our advantage to conserve the reproductive powers of the more valuable dairy and breeding animals, thereby continuing their usefulness as stock getters and milk producers.

Any disorder of the genital apparatus may result in serious economic loss, due to the diminished and irregular supply of milk, and the compulsory fattening of valuable deep-milking cows for the butcher. Acute vaginal catarrh is at present disseminated over wide areas in two continents, and the damage attributed to it is said to be greater than that caused by foot and mouth disease. Many European writers regard it as one of the worst scourges affecting dairy cattle. I therefore offer no apology for bringing the disease under the notice of members of The National.

My attention was first drawn to the malady about a year ago, and since then I have had it under observation from a clinical standpoint. It has happened that a considerable number of dairy stock of all ages have passed through my hands while engaged in testing with tuberculin, and in the ordinary routine of practice. About 1800 head of cattle have been available for examination, most of them periodically, and a record has been kept in various herds in regard to (1) age, (2) nature of the lesions (3) sterility and abortion (4) genital soundness.

Sixty per cent. of the females were affected with chronic vaginitis follicularis in varying degree; most of the animals were bred in Essex, while others were brought from different counties to be tested. Not a single case of the acute disease, as described by European writers, has presented itself.

The chronic form is admittedly a comparatively mild and insignificant affection, which, so far as my observations go, bears no distinct relation to sterility co-existing in the same animal. I fear, therefore, that little value can be attached to the data I have collected, unless they serve to draw more attention to sterility arising from other causes.

Synonyms. Infectious vaginal catarrh, vaginitis verrucosa, chronic vaginitis follicularis, vaginitis et metritis follicularis infectiosa (Hess), granular venereal disease (Williams). Moussu (7) calls the vesicular venereal disease "Contagious vaginitis." To avoid confusion in nomenclature, it would be well to restrict the term "contagious" to the disease under consideration.

History. Contagious granular vaginitis appears to have been first observed in Switzerland many years ago. In 1903, the Swiss Veterinary Surgeons' Society instituted a thorough investigation, and later on, under the able direction of Prof. Hess, a report was published replete with valuable information and statistics relating to the extent and the ravages of the plague.

It is widely disseminated throughout various parts of Germany, and in other European countries it has attracted considerable attention for some years past. Williams (5) considers that the malady has probably existed in the United States for a quarter of a century, although it was apparently only recognised as a distinct disease two years ago. There are reports as to existence in Canada, and indeed it would appear to be widely distributed over the North American continent.

In this country the disease has received little or no attention, and no observations, so far as I am aware, have been recorded in our literature. This may be due to the fact that the acute vaginal and uterine inflammation is a rare complaint, although it prevails as a virulent epizootic and enzootic in other parts of the world.

Although details are lacking in regard to the incidence of the disease, it is reasonable to suppose that English cattle possess no immunity against the acute infective virus. Its non-recognition by English veterinarians may be ascribed to (1) the mild, chronic, and somewhat insidious course, (2) confusion with the vesicular disease known as "bull burnt or clap" (3) the somewhat scant attention paid to sterility in cattle, (4) the prevalence of epizootic abortion due to Bang's bacillus.

ETIOLOGY.

In 1898, Ostertag claimed to have discovered the specific organism in a short streptococcus consisting of 6—9 cocci, held together in a delicate capsule, and present in the purulent discharge and in the deeper layers of the mucous membrane. He isolated the organism, grew it in pure culture, and produced the disease in heifers by inoculation.

It is interesting to note that swabs soaked in both mixed and pure cultures of other cocci associated with the streptococcus in the vaginal discharges gave negative results.

Ostertag went further than this; he pointed out that bacterial invasion of the uterus may take place, in which case the granules are found on the uterine mucous membrane, and the discharge from the uterus is rich in the streptococci. Ostertag's statements were confirmed by Hecker, Raebiger, Hess, Raedzir and other investigators. Raebiger (8), who began his researches in 1900, considered that the streptococcus was the causal agent for the following reasons:—(1) It is always to be found in the diseased secretion. (2) Its artificial cultivations at once produced the typical catarrh. (3) The streptococci are always to be found in artificially infected cows. The organism is said to be non-pathogenic for animals other than the ox. It stains well with Loeffler or methylene-blue, but is decolorised by Gram. It grows well at ordinary temperatures on glycerine-agar, urine-agar, and other media. While infection of the uterus is easy to understand, the fact remains that few authenticated cases have been recorded. In many cases of chronic metritis and pyometra, it is not at all clear that a specific organism, associated with granular lesions on the uterine mucosa, was present in the purulent discharge.

It is true various writers have reported isolated cases of uterine infection. It will, I think, be generally admitted that the diseases of the genital organs, more especially those of an infectious nature, require further investigation. The relation between the granular disease and epizootic abortion has not been proved, although many outbreaks on the continent and in America have been attributed to contagious granular vaginitis. Epizootic abortion is very largely due to the bacillus of Bang, which many European authorities have, so far, been slow to recognise. It may be observed that Ostertag's organism is not universally accepted as the causal agent of contagious granular catarrh. The bacterial contents of a purulent vaginal discharge is large, and several organisms have been isolated by different observers. Prowazek's bodies, which may possibly be protozoa, are said by Blaha (14) to be invariably present in scrapings from the follicular nodules.

Methods of Infection. The malady is described as a venereal disease in that is often transmitted by the coition; other factors in spreading the infection are: Immediate contact (soiled tail, hind parts and channel); soiled fodder and litter, hands, boots and clothes of attendants; grooming utensils (curry-comb), and infection from the mother during and after parturition. The quantity and virulence of the vaginal discharge are in direct proportion to the intensity of the lesions. In young females, inoculation with virulent material is nearly always attended with positive results, the discharge is scooped up from the vagina of a diseased animal and rubbed on the mucosa of a healthy one, or sterile swabs of cotton wool on wire are used to convey the infection. Bulls and old cows are less susceptible to artificial infection, although bulls readily contract the disease by coition. I have examined large herds of cows without conveying the disease from one to another, although no precautions have been taken; in most cases the vulvar labia have been grasped with the thumb and fingers, but a wire dilator or a speculum has also been used with impunity, even in cases showing a slight discharge.

The chronic stage of the nodular disease is very seldom accompanied by a visible vulvar discharge; but it may occur after copulation. I have convinced myself that the common chronic form, unattended by redness, swelling, or increased secretion, may be regarded as non-infectious Hess's (3) observations on this point are very important; he says that "the activity of the virus disappears with the onset of chronicity." In about 2 per cent. of cases unconnected with copulation I have observed a small quantity of pus on the vulvo-vaginal mucous membrane in the form of scanty greyish-white sheds or flakes; at other times a little pus has issued from the clitoris or has been squeezed out from below. This discharge has not, however, proved infective through the bull or in other ways, and the natural assumption is that it has no virulence.

Age. The disease is most intense in young animals in all stages of the catarrh; they are also most susceptible to natural infection. Three-fourths of my cases have occurred amongst young cows and heifers, and a large number of young calves and virgin heifers have shown distinct granular lesions. In old cows, the nodules are usually blanched and few in number or absent altogether.

Doubtless, the tough (pale yellow) condition of the genital mucosa in old cows renders it less liable to infection: while there is a constant tendency on the part of the granules to shrink and fade away, resulting in spontaneous recovery, the process may take several years.

It is reasonable to assume that old cows may acquire immunity from one or several attacks of the acute disease.

Prevalence.—The disease has appeared in several countries in a severe enzootic and epizootic form. In East Prussia 30,000 cows are reported to have been affected in one year; in Switzerland in

1903-1904 60 per cent. of the cows and 48 per cent. of the bulls were found to be diseased. In other countries, in certain areas, it has not been possible to find a healthy herd of cows. In the mild chronic form of the malady it is common in my experience to find 80 per cent. of dairy cows showing distinct to very slight lesions.

Incubative Period.—This depends on the activity of the virus. Inflammatory phenomena begin to appear two to three days after artificial inoculation, three to five days after coition. Two to six days is the usual period. Hess puts the interval at 20 to 72 hours, but records one case in which a delay of eleven days occurred.

SYMPTOMS AND COURSE.

I. Acute Form.—During the first 48 hours the disease cannot be distinguished from other forms of vaginitis. There is swelling of the vulvar labia, increased mucus, and considerable congestion and swelling of the vulvo-vaginal mucous membrane. The cow evinces pain on manipulation, urinates frequently and scantily, strains and arches her back a little, and shows uneasy movements of the tail and hind legs. Very soon the discharge becomes muco-purulent and more or less copious; it adheres to the tail and the tuft of hairs at the lower commissure, and it is found on the floor of the stall; it is always odourless. The sticky nature of the mucus causes the pus to cling to the mucosa in flaky shreds; hence, on opening up the vagina a whitish-grey shreddy coating is found lining the canal.

In about 48 hours numerous small firm rounded elevations appear in the vestibule in the region of the clitoris on the infero-lateral wall. These are the granules which are so characteristic of the disease in all its stages; they are always to be seen and felt under ordinary circumstances, and the diagnosis depends essentially on their presence. They present the following characters:—rounded like mustard seeds, pin's head to a radish seed in size (millet size common) dark red in colour, solid, smooth surface, discrete, and often arranged in irregular parallel rows on the longitudinal ridges of the mucous membrane.

They spread upwards over the lateral walls till they meet near the superior vulvar commissure. Occasionally they spread forward to the os, and even to the uterus. The irritation and discharge bear a distinct relation to the number of granules and the area affected.

It is important to remember that no vesicles, pustules or sores of any kind appear on the mucous membrane in uncomplicated cases. The constitutional symptoms are slight; there may be a little fever, and the milk yield is diminished. The fatality is nil. The acute stage lasts a month or less.

II. Sub-Acute Form. The inflammation subsides and the discharge becomes slight and intermittent. Angry zones or areola still surround the nodules which are now bright red in colour. This stage may last two months.

III. Chronic Form. The chronic stage may be said to set in about three months after the commencement of the disease. Its duration is quite indefinite; there is no doubt that the lesions may and do persist for several years, though there is a constant tendency for the granules to dwindle and fade; they may disappear in a few months.

According to my observations the character of the lesions may vary considerably in the same individual at different periods. Again, the granular areas still persist in the great majority of the cows which were recognised as being diseased twelve months ago.

The symptoms are, practically, those of the acute form minus the inflammatory phenomena. Discharge and signs of irritation are usually absent, the granules, for the most part, retain their former character and situation; they are usually pale red in colour and the surrounding mucosa is quite normal, or occasionally injected and slightly reddened. No catarrh is present. Later on the nodules become blanched or colourless, like little blisters, and smaller in size. Not infrequently large blanched granules are met with on the upper wall close to the vulvar commissure. Different stages are met with in the same animal. The rowed arrangement is less constant after a time, and contiguous granules sometimes become confluent, forming elongated swellings in length from a canary seed up to ⅜in. Rounded swellings are also seen the size of a hemp seed to half a split pea on the surface.

The granules show little tendency to run together in calves and young heifers; in the latter they are often rather large, discrete and bright red in colour. Occasionally they are heaped up round the clitoris like a raspberry. On the roof of the vulva the nodules are usually few in number, small, fade more or less rapidly, and are situate close to the upper commissure ⅛in. to ¼in. from the cutaneous covering; inferiorly the mucosa is free and smooth for ½ to ¾ inch from the margin of the labia.

It follows that the granular ring encircling the tube of the vulva is considerably broader below than above, *i.e.* the granules extend further forward infero-laterally. Below, the breadth is commonly 1½ inches in cows, above it is ½-inch. One frequently finds a few small pin-head blanched granules near the upper commissure (less often two or three larger ones), and an area of discrete bright or pale red mustard seed sized granules on each side and in front of the clitoris.

I have met with several cases in heifers in which the lesions reached the neighbourhood of the os uteri along the floor of the vagina, in one case only they had spread along the roof near the os, while inferiorly they barely reached the meatus. I am inclined to regard this condition as of rare occurrence in the chronic form of the disease. It is certainly the case that the nodules show a more marked tendency to spread forward in heifers than in older animals.

The colour of the mucous membrane varies greatly, it is often very pale or blanched in young calves, pale yellow in old cows, the yellowish tint being quite normal. A diffused or streaky redness is associated with extensive lesions.

Exacerbations occur occasionally during pregnancy more often at œstral periods, especially if copulation takes place. The irritation induced by coition is shown by (1) angry dark red colour of the granular area and surrounding mucosa and slight swelling ; (2 excessive mucus ; (3) occasional tearing at the upper commissure (heifers) ; (4) slight coating of shreddy pus or thick sticky mucus, or both. The slight signs of uneasiness, if present, are unnoticed by the cowmen, who remain in ignorance of the condition until it is pointed out to them. These untoward symptoms notwithstanding, the animal frequently proves in calf.

For some time before and after parturition and abortion, the swollen œdematous slimy condition of the genital canal and labia renders the detection of the granules difficult or impossible. The period varies from a few days to three weeks after parturition, and for two to three up to 12 weeks in primiparæ beforehand. The nodular elevations are partially or completely buried in the swollen mucosa, in spite of which they may be felt with the fingers or even seen occasionally on stretching the labia.

It follows that a definite diagnosis cannot always be made in down-calvers and in recently calved cows.

Mention has been made of the fact that a small quantity of pus may now and then be squeezed out of the clitoris ; this pus, however, possesses no virulence, it quickly disappears and its presence remains undetected unless periodic examinations of affected animals are made. Lastly, petechiæ are very occasionally seen on the vulvo-vaginal mucous membrane at all ages from six or nine months upwards.

Ladanyi (10) has described an exceptional form of the disease in which the typical granules are absent ; instead minute yellowish-white "points" or "spots" appear on the vaginal mucous membrane for a short time only.

CASES OF CHRONIC GRANULAR VAGINITIS AGGRAVATED BY COPULATION.

Cow (*a*), aged four. I examined her October 9th, 1909, three weeks post partum. Numerous pale red nodules size of mustard seed arranged in rows and extending forwards for two inches infero-laterally ; mucosa otherwise normal. December 21st, 1909. Six days after first service, granules dark red and apparently increased in number, mucosa injected, especially upper wall ; lot of mucus, slight coating of pus on mucous membrane, half tea spoonful squeezed from prepuce of clitoris which was swollen. Swabs from this and other cows sent away for experimental inoculation gave negative results: staphylococci and streptococci in cover glass preparations. Irritation disappeared within 10 days; cow pregnant.

Cow (*b*) Aged 3. Similar to (*a*) but granular area smaller before and less irritated after copulation. No pus from clitoris, no swelling; stood bull.

Cow (*c*) Aged 6. Granular area, rather larger than (*a*) but more faded, several canary seed to half split-pea reddish elevations near clitoris. Vulva relaxed and moist. After copulation: granules increased, bright red in colour, surrounding mucosa congested and little swollen, increased mucus, no pus. This cow was sterile, right ovary cystic, size of pullet's egg, œstrum excessive and prolonged, periods regular, becoming shorter and irregular; broad ligaments relaxed, pronounced hollow right side; crushed cysts four times per vaginam, after which she became pregnant.

In both (*b*) and (*c*) the irritation produced by the bull quickly disappeared.

Bulls. The symptoms in bulls are comparatively mild, even in the acute form of the catarrh; granules are seldom to be detected on the glans penis. In Switzerland 48 per cent. of the bulls were diseased during 1903-1904, but only one-fourth of these showed acute symptoms; the others were chronically affected. Bulls are credited with transmitting the disease without showing any visible discharge or swelling.

A purulent catarrh of the prepuce is sometimes present, a little pus oozes from or may be pressed out of the sheath, it may adhere to and become dried on the long preputial hairs. Less frequently, the purulent or muco-purulent discharge is abundant, the prepuce is swollen and very sensitive, and granules similar to those seen in cows are found on the glans penis. The animal may refuse to copulate, or the act is performed slowly and a little blood is noticed.

I have handled about 60 bulls in the course of my investigations, and in no single instance have I found lesions attributable to the granular disease. The great majority were healthy, two or three were affected with "clap," whilst others, mostly young bulls in use for the first time, showed warty growths on the penis or injuries due to copulation. It must be admitted that the cursory examination made in some cases may have failed to reveal slight lesions in spite of the fact that observations were made in most instances before, during, and after coition.

Some of these bulls belonged to herds in which 80 per cent. of the cows and heifers were chronically affected, yet they continued to serve healthy and diseased alike without conveying the infection.

It would appear, therefore, that the symptoms in bulls in the chronic form are negative or very slight, and further, that this form of the malady is not venereal in the proper sense of the term.

The clinical diagnosis of a suspicious genital discharge in bulls may, in some instances, prove rather difficult; in cows, on the other hand, differentiation is usually quite simple.

The fact should be borne in mind that a profuse preputial purulent discharge in the bull is often due to a totally different disease, commonly termed "Bull burnt." Like the granular disease, this affection is sometimes transmitted in other ways than by coition, and pregnant animals may become infected if brought in contact with the virulent discharge.

DIFFERENTIAL DIAGNOSIS.

The scanty literature at our disposal in regard to venereal diseases of cattle is regrettable in that it may lead to confusion in their differentiation. The literature is not only scanty, some of it, especially that of older writers, is positively misleading, *e.g.*, the etiology and differential symptoms of so-called bovine gonorrhœa have not received the attention they deserve.

It is highly important that the two infectious genital diseases, vesicular vaginitis or "cow-clap," and contagious granular vaginitis should be clearly distinguished. I am of opinion that the differential diagnosis in the cow is difficult only (1) during the first two or three days, before the pathognomonic granules appear in the latter disease; (2) when both diseases affect the same animal simultaneously. My experience has been that they may easily co-exist in the same animal; the same remark applies to gangrenous and other forms of vaginitis which need not be detailed here.

At one time (9) the idea gained ground that the granular areas on the vaginal mucous membrane were in reality the result of an acute (vesicular) vaginitis which had assumed a chronic form. This statement, the fallacy of which has been proved by clinical observation, shows the amount of confusion which has existed.

VESICULAR VAGINITIS.

Synonyms. Eruptive or Benign venereal disease, vesicular exanthema.

This is a mild and fairly common affection in cattle which cannot be distinguished from what is ordinarily known as gonorrhœa, bull-burnt, or cow-clap. Rightly or wrongly I have been accustomed to regard it as the form of gonorrhœa affecting cattle. A series of experimental inoculations on a large scale, with a sufficient number of controls, would afford valuable evidence for or against this hypothesis.

In my cases a distinct urethritis has been present in male animals, and in females the meatus has been frequently involved. The late Prof. Walley in his lectures defined gonorrhœa as a specific urethritis in males or vaginitis in females, the positive symptom being the formation of a crop of pustules followed by ulcers on the mucosa.

In the vesicular disease the ejection of urine is distinctly irritating to the animal, it is voided very frequently in small quantities, the straining, arching of the back, elevation and whisking of the tail, together with the discharge of muco-pus from the urethra in bulls, all point to the urethra being involved in the inflammatory process. Accompanying the ordinary symptoms of vaginitis, there is at the outset a vesicular eruption on the mucosa; the vesicles, which are rarely observed in the natural infection, are rapidly followed by crops of pustules and slight superficial sores or ulcers. The genital canal is lined with a greyish-white flaky coating of slimy pus, beneath which the mucous membrane is intensely injected, angry-looking, and swollen. It bleeds readily on examination.

In bulls there is a purulent catarrh of the prepuce, or balanitis, associated with swelling, tenderness, stiffness, and more or less abundant discharge. The penis is also inflamed and ulcers may be observed. Copulation is distinctly painful and draws attention to the condition, there is usually some blood during the act.

Spontaneous and complete recovery often occurs in cows in about three weeks, though proper treatment is advisable to shorten the duration of the malady and to guard against its sequelæ. The inflammatory phenomena may disappear after a few days.

The vesicular disease resembles the acute granular disease in the following respects: (1) inflammatory phenomena (2) incubative period, (3) method of infection, (4) one attack confers no immunity.

It differs in that (1) its duration is much shorter, granules never appear at any stage if the disease exists alone, (2) it is widely distributed in Great Britain, and it is said to be communicable to other animals by experimental inoculation, the granular affection is not. In a word the chronic course of the granular disease and the absence of pustules and ulcers serve to differentiate it.

Several outbreaks of the vesicular-gonorrhœal disease have come under my observation, in females especially they have been quite mild in character; the cows gave less milk for a few days, but most of those infected by coition proved in calf. There was very little rise in temperature. The bulls showed more fever, loss of appetite, and stiffness, and they did not recover so quickly, due, in my opinion, to the urethra being involved.

The following cases of the double infection are interesting:

CASES OF CHRONIC GRANULAR DISEASE INFECTED WITH "CLAP."

Herd A. Consisting of 57 dairy cows and heifers (1st calf), and two bulls. One of the bulls was young, and had not yet come into use. The herd had been under observation for eight months for granular disease; 29 of the younger animals, average age four years, were rather extensively affected, others showed slight lesions, some very slight indeed. Total number affected, 70 per cent. Examined 26 Feb. 1910, found old bull and three cows with swollen genitals and a mucopurulent discharge.

Cow I. Five years. Bulled 21st, swelled 24th, discharged 25th, on the 26th vulva much swollen and œdematous, egg-cupful of yellowish-white slimy pus in channel behind cow. Granules rather obscured by discharge and swollen congested condition of mucous membrane, only to reappear as prominent as ever as the inflammation quickly abated. Raw ulcerated condition of patches of the mucosa, covered with shreddy pus, and bleeds readily. The granular area before the fresh infection covered most of the vulvar mucous membrane and extended half way towards the os uteri along the floor of the vagina. She was temporarily sterile, due to cysts in right ovary; œstrum was excessive (mild nymphomania) and irregular. The cysts were crushed some three weeks later, the operation being repeated for the third time within 28 days. At the second subsequent (normal) œstrum she went to bull, and became pregnant.

Cow II. Three years. Granules mostly bright red in colour and size of mustard seed, confined to vestibule. Bulled 25th. On 26th mucosa little hyperæmic, excess of mucus. 27th, labia little swollen, granules quite distinct, signs of irritation during micturition. 29th, symptoms as above, but less acute. This heifer calved six months ago, and had not been in œstrum; previous examination had disclosed two yellow bodies the size of a winter bean in the left ovary. These were pressed out per vaginam in three weeks' time, with the result that œstrum appeared within 10 days. The corpora formed again, but did not recur after a second operation; normal œstrum and conception occurred in 21 days after second heat.

Cow III. Two and half years. Three months calved, previous examinations showed her to be free of chronic granular vaginitis, hence she may be regarded as a control. Bulled 23rd Feb., 1910. Examined 26th. Characteristic symptoms, already noted, of vesicular disease present, most acute sixth day. No granules present before or afterwards. The genital canal was covered with dirty-white pus almost like a false membrane, beneath which the mucosa was intensely congested and ulcerated. Pain in micturition was shown by dribbling of urine, straining, paddling of hind legs, elevation and uneasy movements of the tail. The internal genitalia of this heifer were normal, and she conceived at the first service in spite of the infection.

Bull. Examined 26th. Showed marked symptoms already described of inflammation of prepuce and penis, with ulceration. Temperature 103.6. Appetite rather poor, thin yellowish discharge becoming thick and nearly white or creamy. Painful micturition. This bull had served six other cows previous to infecting No. 1. On the 19th he had two cows, one belonged to the herd and was rather severely affected with chronic granular disease prior to copulation; the genital mucosa was a little injected and moist for some days afterwards, nevertheless she proved in calf. The other cow was a strange one from a distance. There is little doubt she brought the infection on the farm, though I was unable to verify the fact.

I was able to keep the cows under observation without adopting any treatment; the bull, however, had the sheath irrigated twice daily with $\frac{3}{4}$ per cent. of bacillol and pessaries containing six per cent. bacillol were inserted every night. He was shown a cow in season after four weeks, at which time he seemed all right. Two weeks later he was used with impunity.

Remarks. Before seeing cow iii. and making a careful examination of the bull, I came to the conclusion that I had at last come across a genuine outbreak of acute granular vaginitis. It seemed to me that the home cow served on the 19th had infected the bull acutely. The ulcerated condition of the bull's penis together with the absence of granular lesions in cow No. iii. showed this hastily-formed opinion to be erroneous. I might mention, also, that whilst engaged in testing this herd for tuberculosis I conveyed the infection from the bull to a genitally sound cow and to a heifer, both contracted the vesicular disease like No. iii. but in a milder form. Recovery occurred in a few days. The heifer was pregnant and carried her calf the full term. Other outbreaks of the vesicular-gonorrhœal disease have occurred on other farms at somewhat long intervals during the last few years. The same observations apply to them except that the granular lesions, if present, were not looked for and were not observed. In one large farm (94 cows) the same bull served seven cows at intervals of a day or two and infected every one of them. They all proved to be pregnant. Lysol in half per cent. solution was used to irrigate the parts.

THERAPEUTICS.

Contagious granular vaginitis is to be handled as an infectious disease on the usual lines, disinfection and isolation being essential for its control and eradication. The earlier stages of the malady call for curative and prophylactic measures somewhat similar to those recommended for epizootic abortion: (1) antiseptic irrigations of all animals, diseased and healthy; (2) cleansing and disinfection of the external genitals, tail and hind quarters; (3) disinfection of the cowsheds or other infected buildings, especially floors and gutters or channels; (4) isolation of diseased, suspected or newly arrived animals; (5) cessation from breeding for a reasonable time, six to eight weeks, (three months after abortion).

The later or chronic stages do not appear to require any treatment. Raebiger (8) carried out classic experiments from 1900 to 1906; after exhaustive investigations he found that a creosote preparation named bacillol most nearly fulfilled the requirements of a reliable, cheap, and (comparatively) non-irritating antiseptic for irrigation purposes. 1½ per cent. solution killed the streptococci in one minute, while strong solutions proved less toxic than other preparations. Personally, I have used bacillol largely in ¾ per cent. solution for both vaginal and uterine irrigation with excellent results. Lysol is more soapy in character, and is to be preferred, but it is too expensive for general use. Two per cent. solutions of these disinfectants, together with creolin and a host of coal tar derivatives, are too strong for tender, sensitive genital mucous membranes, both male and female; I have repeatedly observed, more especially in young animals, irritation and straining with tendency to adhesions or stricture from solutions of this strength employed twice daily for two or three weeks. Williams' (5) researches emphasise this fact, and show that irritation produced in this way interferes considerably with subsequent more rational treatment. The harm sometimes done by strong injections is also referred to by Hess. (3A)

In dealing with a small number of cows, thorough flushing out of the vagina is most conveniently effected by means of a hard metal syringe holding about one quart, strongly made for rough usage, the nozzle or injection pipe six inches long, and thick to avoid injury to the vagina or os from careless holding, the distal end slightly bulbous and pierced with numerous holes so as to produce a fine spray in every direction without too much force. For bulls, an extra long Higginson's enema or milk fever syringe with special metal nozzle, or the following appliances used for cows may be employed. Instead of the special vaginal syringe a gravity apparatus is used consisting of a large funnel, several feet of rubber tubing with a canula attached. A rubber tube ten feet in length ⅜in. bore and ¾in. outside diameter is to be recommended for both vaginal and uterine irrigation used with a funnel, attached to any enema pump, a pail or tank fitted with a stopcock, or with a sufficiently large tube holder and suction plate to keep it down and prevent kinking. It is sometimes possible to suspend a large pail above and behind the cows and to pull it along the shed ; this facilitates the work very much in large herds and economises the cowmen's time.

The irrigations with mild warm solutions are carried out twice daily for a few days, then once daily, becoming less frequent after two to three weeks. After washing out the whole vaginal canal, bacillol ointment six to ten per cent. is used once daily for a time in capsule or pencil form ; the pessary is best introduced as far as possible with the fingers. This is Ritzer's method of using the ointment ; Raebiger has invented a special ointment syringe for the purpose. The pencils are cheapest and they give good results in other vaginal affections.

It is highly necessary to prevent the animals copulating till all inflammatory phenomena have disappeared, and for a short time afterwards, roughly six weeks. The irritation sometimes produced by coition, even in the chronic disease, has already been alluded to. Recovery is retarded, and infection may be conveyed in this way. It is good practice to syringe out the vagina with a one per cent. solution of bicarbonate of soda before going to bull, in case an acid or neutral state of the mucus exists ; the reaction should be feebly alkaline, and various conditions of the genital tract may alter this.

Little more need be said in regard to prophylaxis. If the sound animals are few in number, and other premises are not available, they should occupy stalls apart from the other cows to which the drainage does not flow, *i.e.* at the higher end of the shed. The sound cows should be dressed first, and all fresh arrivals disinfected and kept under observation. A garden syringe is useful for spraying the hind parts after cleansing the soiled vulva and tail.

It is worthy of note that recovery is not necessarily associated with the disappearance of the granules, though they often do disappear if prompt and rational treatment is adopted. If they persist they are small, blanched, and probably few in number.

As an experiment, I applied the bacillol treatment to one rather severely affected herd for four weeks as follows:—

First week—Daily injections $\frac{3}{4}$ per cent. solution and pencil ointment six per cent.

Second week—Daily injections, pencils every second day.

Third week—Injections and pencils three times per week.

Fourth week—Two injections only.

Result—No irritation, distinct fading (loss of bright red colour), of granules in a number of young cows, probably fewer in number. The older cows showed little alteration.

SEQUELÆ OF GRANULAR VAGINITIS—ABORTION AND STERILITY.

We have seen that the disease in the individual is comparatively mild, attended with little constitutional disturbance, and of short duration in the acute stage. Its results, however, according to European investigators are very serious indeed. Many careful observations have been made, particularly by Hess, whose excellent work deserves all praise. It is held that serious economic loss is produced by the extension of the catarrh to the uterus, and that both sterility and abortion are common in severely affected herds. That an infective lesion in one part of the genital canal may extend to another by continuity will be readily admitted, at the same time the number of the uterine infections is not stated, and some writers seem to attribute almost every disease of the genital tract to this malady. Their facts are beyond dispute, the deductions drawn from these facts are, perhaps, open to criticism.

My point is that the granular affection in the virulent form has a place, and an important place, in the production of sterility, and it may be of abortion, but the tendency is to magnify its importance as an etiological factor. Hess (3) states the case very fairly when he says:—"It is not a matter of doubt that lack of desire and impotence, as well as some ovarian and uterine diseases, a cystic degeneration of the ovaries, mortification and maceration of the fœtus, abortion, chronic catarrhal and purulent endometritis, occur equally in infected and non-infected genital organs, and declare themselves by quite similar symptoms, on which account their etiological significance is at times surrounded with great difficulty."

ABORTION.

The relative importance of contagious granular vaginitis in the production of abortion has been variously estimated, and probably over-estimated. It is certainly the case that a purulent catarrh of the uterus, secondary to specific lesions in the vagina, or dating from the last parturition, destroys the intimate union which exists between mother and fœtus, and causes the latter to be expelled as a foreign body.

Invasion of the uterus is most likely to occur during œstrum through the open cervical canal: if impregnation takes place abortion will very probably take place during the first few weeks of pregnancy. An endometritis may already exist, and conception may possibly take place, in which case the fertilised ovum is unlikely to have a long intra-uterine life.

In discussing the etiology of granular vaginitis, I ventured the statement that the number of authenticated cases recorded of granular uterine infection is small; it now appears that the number of abortions attributed to it is very large. Many European writers are, or have been of opinion that it is responsible for much of the abortion which prevails in an epizootic form. If the nodular disease is mild, the latter is small or *nil*; on the other hand if the nodular disease is severe, it is credited with producing all the abortion in the affected herds amounting to 20 to 70 per cent. of the total number of cows. At the same time it is admitted that in other severely affected herds, no abortions at all have occurred.

One is struck by the lack of information in regard to other possible causes. To establish the above hypothesis it seems to me that three things are necessary:—(1) General recognition by bacteriologists of Ostertag's streptococcus or some other organism as the causal agent; (2) the demonstration of the specific organism and the granules in the uterus; (3) the transmission of the disease to other animals by experimental inoculation.

It is hardly necessary to say before a gathering of English Veterinarians that by far the largest number of abortions in this country are due to Bang's bacillus of cattle abortion. The identity of the English and Danish disease has been established by the Departmental Committee of the Board of Agriculture. It is natural to suppose, therefore, that much of the epizootic abortion which prevails on the continent and in America may be due to the bacillus of Bang, in spite of the fact that this organism has so far received scant attention as an etiological factor.

In my own practice I have kept a record of 300 consecutive cases of abortion: 80 per cent. of these have been clearly due to Bang's bacillus, judging by (1) character of the exudate; (2) microscopic examination of fœtal cotyledons; (3) appearance of the after birth; (4) history and number of cases in the herd. The average period of gestation was six to six-and-half months, and the average age four-and-half years. All these cows were sent to isolation farms, most of them when they were showing premonitory symptoms, and their arrival was notified to me in every instance.

It is a well known fact that epizootic abortion (Bang) is largely a disease of heifers and young cows, the same applies to the abortion produced by the granular disease. The latter, however, occurs at any period, often within 12 weeks of fecundation, whereas most cases of epizootic abortion occur between the 5th and 7th months of pregnancy.

I have not found severe granular lesions in slipped cows or heifers, although a number of these have been examined at various periods both before and after the act of abortion. On the contrary, the signs of chronic granular vaginitis have been comparatively slight and insignificant, although a chronic purulent endometritis leading to temporary sterility has been a common sequel of the abortion. Moreover, I have not found any relation between the extent of the lesions and the number of abortions, in either freshly-infected or older-infected herds.

STERILITY.

It is generally accepted that sterility stands in intimate relation to the evolution of the milch cow and the forcing methods adopted at the present day. It certainly appears to be on the increase in this country.

While sterility occurs without infection of the genital organs, it is considered to be closely related to and to follow severe attacks of contagious granular vaginitis. Many European investigators and writers agree on this point, *e.g.*, Zschokke, Raebiger, Hess, Hutyra and Marek, Neilson, Ostertag and Thoms. Their observations and statistics show that a much larger number of ovarian and uterine diseases are met with in severely infected herds, and, further, that the number of sterile animals affected with granular disease is considerably greater than the number of non-infected animals.

At the Ninth International Veterinary Congress at the Hague, on September 19, 1909, Hess (13) stated that so far as his observations went, out of every 100 cases of sterility in cows, 58 were due to this disease. He (3) points to infected herds in which the pregnancy figure is only 20 per cent. Hess also considers that apart from the extension of the catarrh to the uterus, the protracted hyperæsthesia of the posterior genital tract in itself hinders conception.

In other words, a chronic irritation at one end of the genital canal is said to inhibit ovulation and fertilization at the other, or to produce a definite pathological lesion such as a cyst or an enlarged corpus luteum in the ovary. It is difficult to understand how this may happen. If the vaginal mucus gave an acid reaction non-conception would be easily explainable, but this apparently is not the case.

The diseases of the genital organs attributed to the granular catarrh need not detain us here, and I propose to do little more than enumerate those most frequently met with: ovarian cysts, persistent or hypertrophied corpora lutea, occlusion or thickening of the oviducts, metritis and endometritis in various forms with or without pyometra, thickening, occlusion, and alteration in shape of the cervix. These diseases produce sterility in various ways, many of them are the direct result of normal parturition, or abortion with retained placenta, or faulty involution of the uterus. If conception occurs, death and maceration of the fœtus may ensue followed by a muco-purulent discharge, and possibly abortion or pyometra; on the other hand, the fœtus is sometimes carried full-time or over-time. Abnormal œstrum usually accompanies sterility, there is extra heat or very slight œstrum, irregular periods, nymphomania, or absence of œstrum ("dumb bullers").

In Switzerland (11) a record was kept of 300 cows in which the cause of sterility was definitely ascertained. 173 or 58 per cent. suffered from infectious granular catarrh (only 11 acutely), while 127 were free of the disease. Of the infectious cases 75 per cent. of the 173 cows were between three and six years old; 91 or 57 per cent. had diseased ovaries, either cystic degeneration or hypertrophied yellow bodies; the remainder, 82 in number, gave the following results: endometritis 46 (24 due to abortion), pyometra 21, absence of œstrum 6, acute granular lesions 11.

It is instructive to compare these figures with those pertaining to the 127 sterile cows unaffected with granular disease. 73 per cent. showed ovarian disease (against 57) and 62 per cent. of these had cysts (against 42).

Hess's observations show that, in Switzerland at any rate, ovarian disease is common and that it is primary; endometritis is not often associated with it.

Albrechtsen, on the other hand, takes the opposite view, both in regard to the relative frequency of uterine and ovarian diseases, and the influence of infectious vaginal catarrh on the genital organs. This observer lays stress on the frequency of uterine ailments as a cause of sterility; he regards the endometritis as primary and the cyst secondary. According to Albrechtsen (4) "infectious forms of vaginitis play a proportionately small *rôle* as causes of sterility in the cow; acute vaginitis, with severe symptoms, is rather easy to cure, and seldom causes sterility. Chronic vaginitis follicularis is a very harmless affection which bears no causal relation to co-existent sterility."

A small minority of observers point to herds in which the sterility figure was unaffected by the disease. Ladanyi (10) states "In Hungary, where the disease was very severe, there was no decline in the stock, and sterility was no more frequent than usual." The Swiss veterinary surgeons in the Report already referred to were also not quite unanimous on this point.

My own observations are confined to the mild chronic manifestation of the disease, which, as we have seen, is an insignificant and comparatively speaking harmless affection. I began my examinations in dairy herds in which the sterility figure, both relative and absolute, was high—10 to 20 per cent. Many of them were young animals, the ovaries were largely cystic, and granular lesions, usually more intense in the heifers, were present in 80 per cent. of the females. The internal genitalia of the non-pregnant animals in each herd, examined per rectum, revealed a considerable amount of disease, mostly in the ovaries and the uterus. In other herds, in which the vaginal mucosa alone was observed, the number of affected animals averaged 60 per cent., the lesions were, on the whole, very insignificant, and the sterility figure varied from 1 to 6 per cent.

These early examinations, together with a perusal of the literature on the subject, led me to believe that *the prevalence of sterility in a herd bore a distinct relation to the intensity of the granular lesions, and to the percentage of animals affected.*

Further observations in other herds and in other districts did not confirm this opinion, and at the present time I am not at all sure that the mild form of the disease has any appreciable effect on the reproductive organs. My case book shows that 41 cows, average age 5 years, have been handled for sterility during the past year, either operated upon *per rectum* and *per vaginam*, or treated medicinally, or both. Most of these were absolutely sterile, a small number was relatively so, having had the bull four or five times or more without proving in calf, or showing no œstrum for a long period. A number of others were merely examined and afterwards fattened and sold. The 41 females included 12 heifers (one calf).

The ovaries were affected in 55 per cent. of cases, occasionally accompanied by asymmetrical enlargement of the uterus (catarrhal endometritis). The interesting point here is that one-third of the animals showed no granules in the vagina, in the others the granular areas, excepting in some of the heifers, were faded and very slight indeed.

Turning my attention to calves and young heifers, I found a large proportion showing distinct nodules unaccompanied by irritation of the mucous membrane in almost every instance. The proportion of affected calves increased as age advanced, although the percentages varied considerably in different herds approximately the same age.

The method of infection was by no means clear, unless the fodder or bedding carried the virus, the stockmen did not enter the cowsheds, and in most instances the calves, after weaning, were kept on premises apart from the stock.

The following figures relate to 120 calves, they are rough averages:—

Two to three weeks to four months,	10 to 30 per cent. affected.
Four months,	40 per cent.
Five to nine months,	50 to 55 per cent.
Twelve to fourteen months,	60 per cent.

Several hundred heifers about 18 months, shortly before going to bull, have given a percentage of 65 to 70.

Cows and heifers after first calf, 80 per cent.

The lesions become less and less apparent as age advances, until, in old cows, they are either absent or very slight, small and blanched. It follows that the disease affects large numbers of females, especially young females, it is chronic from the outset, and it apparently does little or no harm. The fact that the disease is present in heifers before they go to bull is significant. In this connection, Williams (5), who has done valuable work in America, states from personal observation that "sterility is very common and ruinous in herds where the disease is already present in the virgin heifers, and the basis for the sterility is laid prior to the coition." It is interesting to note that small ovarian cysts are not uncommon in virgin heifers that have never been bred, and I have found these to occur equally in infected and non-infected animals. In herds kept under observation, heifers after the first calf usually stood the bull the 1st or 2nd time, the presence of granular lesions in the vagina did not affect conception to any appreciable extent.

In young heifers first time bred, sterility has been still more uncommon, although a case of nymphomania due to cystic ovary, or a "spurious pregnancy" due to pyometra, has occasionally occurred, nor has the so-called "invisible abortion" been at all common during the first few weeks of pregnancy, although at later periods in gestation epizootic abortion (Bang) has caused much havoc in some herds, up to 34 per cent. (12 out of 35 young heifers).

It must be remembered that the effects of the nodular disease may remain after the primary vaginal lesions have faded. Bearing this in mind I have not been able to trace a higher percentage of subsequent sterility in young animals in which the vaginal lesions were extensive.

Twelve months is of course far too short a time in which to form a definite opinion based on personal observation. A series of extended observations in widely separated districts would furnish information of considerable value to breeders and to veterinary surgeons interested in sterility, particularly if statistics as to genital soundness were obtained by rectal examination.

I have made a systematic examination per rectum in a few herds to determine the relative frequency of disease in affected and non-affected animals. It was my original intention to submit these and other data here; they are, however, rather voluminous and hardly germane to this paper, inasmuch as they do not establish a causal relationship between sterility and abortion on the one hand, and chronic granular disease on the other.

In conclusion, I have to submit the following suggestion "That the National Association shall form a committee to ascertain if contagious granular vaginitis actually exists in a virulent form in this country, to note its effects, and to report to the next meeting." If the terms could be extended to include sterility in cattle I feel sure valuable results would ensue.

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Discussion.

Mr. S. Stockman: Mr. Chairman and Gentlemen,—In the first place I wish to thank Mr. Wilson for bringing this subject before us in a paper that provides matter for discussion. At the start I would also like to apologise for want of dogmatic opinions in relation to this disease. I have some convictions, but my attitude is rather that of philosophic doubt in connection with quite a number of matters upon which Mr. Wilson seems to me to have expressed himself fairly definitely. When I agreed to open a discussion on this subject, I had done a few experiments, and I was conversant with a good deal of the literature; and I must say that I thought the problem was comparatively simple on the assumption that the views of our Continental colleagues were correct. What I thought was that all that would have to be done here would be to confirm those views. I have not done a very large number of experiments, but so far as I have gone I have found that it is a more difficult subject than I anticipated. I have not in many respects, so far, been able to confirm the work which has been done on the Continent. There is certainly a contagious vaginitis in this country; that I think will be admitted by most practitioners, and the clinical evidence in favour of it being contagious leaves very little to be desired. From the clinical evidence which practitioners are able to supply, one would accept it, I think, as a contagious disease, but if it be necessary to bring forward experimental evidence, I can only say that I have several times been able, by means of swabs passed from the vagina of a sick to that of a healthy animal, to convey the disease. I do not think that that proof of contagion was necessary in relation to the vaginitis that we have in this country, but I thought it just as well to mention it.

I am inclined to differ from Mr. Wilson in his interpretations of vaginitis. He draws a distinction between what has been called on the Continent and what he calls the granular disease, and the acute form of vaginitis which one meets in this country—I have met with only one form in this country—that which he refers to as acute gonorrhœa. I have had several cases of this acute gonorrhœal affection brought to my laboratory, and one might say that it is characterised by a more or less abundant muco-purulent discharge, and in some cases, though not in all, by the appearance of abrasions or, if you like, superficial ulcers. In two cases which were sent to me from outside, both of which were said to have been infected by one bull, I found in one case distinct ulcers which extended up the urethra to the bladder, and I also found them on the external genitals. In the other case I could find no ulcers at all, and I may say, because it seems somewhat important in relation to the paper, that in both cases the so-called granules were well developed. In the other cases which I have observed, which have been fairly numerous, I have never had the animals long enough under my care to see whether granules formed or not after the acute stages had passed off.

As regards the bacteriology of the disease, I do not think I am really in a position to give much information. Ostertag has said that it is due to a streptococcus, and others have confirmed his opinion. So far as the disease that I have met with in this country is concerned, I have had no trouble in finding streptococci in the discharges, but I have had trouble—in fact, I have failed—with the cultures of these streptococci to produce the disease. It would be presumptuous on my part to say that Ostertag had made a mistake; he may have been dealing with a different disease: but so far as my enquiry has gone—and I admit it is not very far—I have no evidence whatever that the streptococcus of Ostertag is the cause of the contagious vaginitis which we find in this country.

As regards the granular form, or the so-called granular form, I have had no difficulty whatever in finding granules in the genital passages of cows. My difficulty has been not to find them. In going through the animals in the laboratory to see whether by chance the disease existed, I discovered that a certain number of nodules could be found in nearly every cow that I examined, and my first feeling was that this disease, as on the Continent, is very prevalent, and that most cows had it. Afterwards it seemed to me that these granules possibly were not indications of disease at all, and in looking for animals to experiment on with vaginal material I had practically to select in every case the animals in the laboratory which showed the fewest nodules. Taking swabs from some of the animals that showed most nodules and putting them into the vaginas of other animals which showed only a few—because, as I say, I was unable to get one which showed none whatever—I failed every time to produce any disease. Moreover, in some cases I scarified the vagina and then introduced the swab, and the results were no different. But there was a result of a kind, that is to say, the swabbing and the scarifications produced a certain amount of irritation, and when that irritation was produced it seemed to me—in fact, I am certain—that a greater number of nodules became visible. They faded in a few days, and the interpretation I was inclined, and am still inclined, to put on that was simply that the irritation had made these nodules—which possibly had nothing to do with disease, stand out more prominently and be more apparent. In that connection I would like to turn to Mr. Wilson's paper (page 60) where he says, "I have examined large herds of cows without conveying the disease from one to another, although no precautions have been taken; in most cases the vulvar labia have been grasped with the thumb and fingers, but a wire dilator or a speculum has also been used with impunity, even in cases shewing a slight discharge." The only interpretation that one can put upon that is that it was not a contagious disease he was dealing with. I am quite convinced that if he had been dealing with the disease which

I refer to as vaginitis he would have been certain to spread it by such methods. You can spread it with the thermometer. In one case it was spread to six heifers in my laboratory, and the only origin I could get for it was that their temperatures had been taken with a thermometer which had been used to take the temperature of an animal which had had this acute disease something like four months before.

I would also direct attention to page 61, "the Symptoms and Course," where Mr. Wilson gives us a description of vaginitis which to my mind describes very well the disease with which I have met, as far as I can make out from the description, Mr. Wilson says that "During the first forty-eight hours the disease cannot be distinguished from other forms of vaginitis. There is swelling of the vulvar labia, increased mucus, and considerable congestion and swelling of the vulvo-vaginal mucous membrane," and so on. Then I gather—he will correct me if I am wrong—that if after these symptoms he does not find granules he concludes that the disease is gonorrhœa. If he finds granules he concludes that the disease is granular vaginitis which has been ushered in by an acute stage. I venture to submit that that is hardly a sufficient argument upon which to base a definite distinction between two diseases.

I propose to pass over the part of the paper dealing with therapeutics—I have had no particular opportunity of trying the effects of therapeutic agents on this disease. I would like to come next to a subject upon which I have rather definite opinions, namely, the relation between vaginitis so-called and abortion. It seems to me that the relation between abortion and vaginitis has been founded upon the suggestion, amongst other things, that a catarrh of the vagina may spread to the uterus and that would be sufficient to cause abortion. Of course, it is difficult to deny that such an event might cause abortion, but the evidence that vaginal catarrh does extend to the pregnant uterus is, I submit, not forthcoming; it has not to my knowledge been established. Other evidence is that granular vaginitis, like abortion, is widespread, and that it co-exists with abortion. Of course, that could be merely a coincidence, and it is not a strong argument upon which to found a connection between vaginitis and abortion. Another argument is that specific abortion which arises from the bacillus of cattle abortion causes an animal to abort in the sixth or seventh month of pregnancy, whilst abortion arising from vaginitis is produced in the fourth to the twelfth week of pregnancy. That cannot be accepted as a distinction. It is quite wrong to suppose that the abortion produced by the abortion bacillus of cattle occurs mainly at the sixth or seventh month. During the enquiry into abortion we have examined a very large number of fœtuses, not only from aborted cows at the laboratory but sent up from the field by practitioners and farmers, and we formed the opinion that cows may abort at any period. Our experimental information is that they may even abort early after infection (a month), and it is no argument whatever against early abortion being due to the bacillus of cattle abortion to say that the abortion has taken place in the first month or three months of pregnancy.

If you examine the available evidence further you will see that the question of contagious abortion has not been eliminated by an examination of the discharge or the membranes by our Continental colleagues, and if you look particularly at Hess's description of how the vaginal disease acts on the uterus you will see that he says it produces a purulent catarrhal state with separation and laceration of the fœtal membranes. That is a very good description--at least it is not a bad description, of a uterus affected with contagious abortion of cattle. He does not say, nor do any of his *confrères* say, that they did eliminate the abortion bacillus; they are dumb, really, on the question of the contagious abortion due to Bang's bacillus.

The next point is the connection between granular vaginitis and sterility. Apparently this granular disease, if it is a disease—I am not prepared to say it is not—is very widespread in this country. If it be the cause of sterility, sterility ought to be equally wide spread, that is to say, we ought to find sterility in this country connected with granular vaginitis. I do not know that any such coexistence has been found, and I gather from what Mr. Wilson says that he is not inclined to believe from his experience that this vaginitis which he has inquired into is a cause of sterility. Hess, who has been responsible for a good deal of the information circulated, states, as Mr. Wilson has quoted, " In other words chronic irritation at one end of the genital canal is said to inhibit ovulation and fertilisation at the other, or to produce a definite pathological lesion, such as a cyst or an enlarged corpus luteum, in the ovary." That is like many of the suggestions about the spiritual world, for instance: it is very difficult to disprove and equally difficult to accept. I do not think it can be taken as evidence at all; it is merely a hypothesis, and I venture to say it is not a particularly good hypothesis. A good deal has been based on the figures which were collected in Switzerland in relation to vaginitis and sterility. The Swiss veterinary surgeons, according to Hess, who brought the figures together, examined in all 4207 females and 115 males. They found 40 per cent of the females healthy and 60 per cent affected with vaginitis—in the granular form, I take it. Of the infected animals he gives particulars of 1893 which had been to the bull, that is to say which had a chance of becoming pregnant, and of the infected he found in-calf 846. He does not exactly say so, but he suggests, that 131 which had aborted should be excluded. I do not think that is warrantable, for they must have been pregnant to have aborted. The total, if you take in the aborted, is 977 which were not sterile; that is 89 per cent. of the whole, and 116 which were sterile; that is 11 per cent. The figures then to my mind would go to prove that vaginitis has nothing to do with sterility.

Of the non-infected he had particulars of 1667. He found in-calf 934 or 56 per cent.; he found non-pregnant 733, 44 per cent. He does not say, however, whether all those 733 had been to the bull and had had a chance of becoming pregnant. I think he implies it, because he does not say they had not, whereas in talking of the infected animals he differentiated between animals which had been to the bull and animals which had not. If it be, then, that these 733 had been to the bull, he found in the non-infected 56 per cent pregnant and 44 per cent not pregnant, showing that the disease of sterility was also very prevalent in animals which had not vaginitis. Of course, it may also be said that the sterility may arise from an infected male. For instance, it might be suggested that the action of the discharge was destructive to the sperm, or that during the act of copulation, the uterus of the cow had been directly infected with the catarrhal disease, but in my limited experience—and I admit it is limited—I have found that several of these animals, which I was in a position to slaughter shortly after the copulation which I considered was the one which infected them, were pregnant. It would appear, then, that infection of the uterus by the bull is not the explanation, and it may be pointed out that the claim really is that vaginitis in the female leads to sterility. It is also said that bulls which have had two or three attacks of disease show a lack of desire and inability to fructify. That may be so, but I would point out that to connect it with vaginitis the disease would have to be proved in connection with each bull, and moreover that the trouble might be mechanical; one is quite prepared to admit that with a chronic inflammation of the urethra a bull might get a stricture which might prevent him from fertilising cows. That, however, would be but an indirect effect of the disease, if it be an effect at all. Moreover, this would not be evidence in favour of the connection between vaginitis and unfruitfulness in the cow It has not come within my experience, but I have repeatedly read, that in some of these herds the same bull will get some animals pregnant and not get others pregnant.

Lastly, I notice in Hess's paper that he says, "Many owners report that after six months' treatment"—that is treatment of vaginitis—"only 40 per cent of the animals get well." This seems to me to mean either that the treatment is not good or that the wrong complaint is being treated, and I do think that one may fairly suggest that in inquiring into sterility one should not neglect the question of whether sterility is a disease by itself which has become confused with vaginitis or with the appearance of granules in the genital passages.

These are the chief remarks that I wished to make upon this paper. I think really the keynote of the whole question is that vaginitis calls for further investigation, and until we have more definite information concerning its pathology it is not very likely that we will arrive at any very definite conclusions regarding its effect upon the genital organs.

Sir John M'Fadyean: I should like, in the first place, to congratulate Mr. Wilson on having produced a paper which is calculated to provoke a good deal of discussion. That may appear to be a left-handed compliment, because I take it the more errors there are in the paper the more discussion there is likely to be about it. I recognise that Mr. Wilson's paper has got some merits, but on the other hand I confess that to me it has been very disappointing, and I feel inclined to condole with him on having had to read a paper about a disease of which he has absolutely never seen a case.

Now that raises a question of what is contagious granular vaginitis? As a distinct disease of cattle this was first differentiated in Germany, and practically all the literature we have on the subject emanates from German and Swiss writers. What is the account which they give of it? In the first place it goes without saying that it is a highly contagious disease: it is called contagious granular vaginitis. Now I want to point out that the disease which Mr. Wilson has been dealing with in these herds to which he refers was by his own account not contagious. He has unconsciously accumulated a great amount of evidence to show that the disease with which he was dealing did not spread naturally. It was not spread from cow to cow by the bull, and he goes out of his way to tell us that he fingered promiscuously the genital organs of diseased and healthy animals, and found he did not spread the disease by infection in that way. Even if that were the only reason for maintaining that Mr. Wilson has not seen a case of contagious vaginitis, it is conclusive, because Mr. Wilson is not entitled to say that contagious granular vaginitis may not be contagious. When we speak of contagious granular vaginitis we are bound to take the account of the German writers on the subject until he or somebody else, from investigation on the spot, can show that these accounts are wrong. It is quite clear that Mr. Wilson did not diagnose this disease because he found that he was dealing with a contagious affection. If I am not mistaken, Mr. Wilson had no notion that he had this disease in these herds until a visit was paid to them, in his company, by Dr. Williams. Now in advance I will anticipate an argument that he may use. He may say that he has Dr. Williams' authority for declaring that the disease in these herds was contagious granular vaginitis, but I had the advantage of a conversation with Dr. Williams before he went to visit these herds, and the fact is that Dr. Williams was himself befogged, and did not know exactly what contagious granular vaginitis is. If my memory does not deceive me, he told me that he had formed an idea that there was not a healthy cow stock in the State of New York. That opinion was not based upon his having found that there were clinical conditions corresponding with those described by the German writers. He was making his diagnosis on the discovery of granules at the side of the clitoris, and I strongly suspect that Dr. Williams has misled Mr. Wilson. He has been diagnosing the disease in animals that were perfectly healthy, or at any rate, if there was any disease at all that disease was not the one described by the German authors. It really is somewhat preposterous to suggest that this disease, which is absolutely of no account from an economical point of view, corresponds with the German disease. How did it become to be known that there was a disease of this sort in Germany? Demands came from the agriculturists to have the disease investigated, because it was spreading to such an extent amongst their herds that it was the cause of widespread sterility. Now, one fault I have to find with Mr. Wilson's paper is that he mixes up in a confusing way his own account of what he saw in these cows in Essex and the description of the German writers. For instance, on page 61, under the heading "Symptoms and Course" I find what is practically a transcript of the German and Swiss account, but as far as I can gather from Mr. Wilson's own account he has never seen this acute form of it at all. The acute form here described is the form which is seen usually within two or three days after a cow has been served by an infected bull. It would have been of enormous interest to us if Mr. Wilson could have given us an account of any observed case in which the disease cropped up in a cow after service by a contaminated bull, but he has not given that, he has got no information of that kind. He speaks about the chronic form of the disease, and I would like to ask him what he means by the chronic form? Does he mean that there are two diseases, one acute and the other chronic, or does he mean merely the chronic stage of an infection which was originally acute? However that may be, Mr. Wilson admits that the bulk of the cases which he saw were in what he called the chronic form; and as I have said before, he has not included in his paper any evidence whatever that the disease was spread by contagion. He has assumed that there was spreading by contagion owing to the fact that he could get 80 per cent. of cows showing these granules on the mucous membrane of the vulva, at the side of the clitoris, but in the absence of evidence that the disease was contagious everyone will decline to admit that as a reliable means of diagnosis. My point is, observe, that the German disease is diagnosed by the fact that it is contagious, and mainly spread by the bull. It is partly diagnosed, I admit, by veterinary surgeons in those countries by the discovery of granules on the vaginal mucous membrane. It might be expected that if this streptococcus described by Ostertag were the proved cause of the disease, one could always determine whether any suspicious case was one of this disease or not by searching for and discovering this streptococcus or proving its absence, but Mr. Wilson does not pretend that he has been able to diagnose his cases and identify them with the German disease by discovering the streptococcus, and I do not hesitate to say that there is no reliable evidence to connect this streptococcus with the German contagious disease. I have not seen it put forward seriously in any German text book that when a doubtful case is presented the person making the examination should arrive at a diagnosis by making a microscopic examination of the vaginal discharge.

One astounding feature of Mr. Wilson's paper is that he not only found 80 per cent of the cows affected with what he diagnoses as contagious granular vaginitis—although there was no evidence of its spread by contagion—but he also found that 60 per cent of the young cattle under 14 months old were diseased, the diagnosis being based on the detection of granules at the side of the clitoris, on the vaginal mucous membrane. But then Mr. Wilson tells us that this disease in the young calves was not the least barrier to conception, that they conceived and held to service just in the same way as animals which showed no granules. I maintain that that is proof positive that the disease which Mr. Wilson saw was not contagious granular vaginitis. I have already said that one is bound to accept the German account of the disease until it has been controverted by somebody making an examination locally; and I put it to Mr. Wilson whether it be not the fact that all those who have dealt with this subject have declared that sterility affects somewhere between 20 and 30 per cent of the cows that become affected with this disease? If that is the rule in Germany is it credible that here in Essex the same disease can exist and yet not cause sterility at all?

The last question I propose to deal with is this—Is there any reason for thinking that the German disease does exist in this country? Well, my answer to that is yes. At any rate I have had brought under my own observation—and you, sir, are acquainted with some of the cases—cases in which the history of the outbreak agreed exactly with the German account; that is to say, in a large herd of cows the introduction of a particular bull had the following consequences. The majority of the animals, both cows and heifers, served by that bull failed to hold. The other animals in the same herd, served by a different bull, held, everyone of them. I ought to have said that immediately after service by this particular bull the cows and heifers had vaginitis, and most of them developed distinct granules at the side of the clitoris; but they went on bulling and could not be got in calf. Now it might be said that these facts do not prove that the sterility was due either to service by that particular bull, or that it was due to the vaginitis, but in some instances a healthy bull put to serve one of these infected cows conveyed the disease to the next healthy cow that he served. That is coincident with the German account of the disease. Although it is stated by most of the German authors that the disease may be spread by other means than the bull, I, for one, am very incredulous about that, because in this particular herd there were plenty of opportunities for indirect infection between the diseased cows and the healthy cows; yet experience showed that unless the healthy cow was served by a contaminated bull she did not get the disease.

I hope Mr. Wilson will take these criticism in good part, because it is very important that we should speak out our minds in these discussions, and although I have had to differ from Mr. Wilson, I think he cannot complain that I have differed from him without giving reasons.

Mr. J. H. Norris: I think this Association is to be congratulated on the subject of this paper, and I think we should be very grateful to Mr. Wilson for bringing forward a paper which contains a good deal of original information, and brings together a rather scanty and ambiguous literature on the subject. I am going to give you my own experience as I have gained it in Ireland. It is a quite recent experience and is rather limited, but in the short time I have had no difficulty whatever in finding these granular lesions in the genital passages of both fruitful and sterile cows, as well as maiden heifers. Most of my observations were made in herds where the sterility figure was very large—considerably over 50 per cent. It is a very strange and remarkable thing that this disease, although it must have been in Great Britain and Ireland for a long time, has received no attention until quite recently. The cause of that, as Mr. Wilson says, may be due to the insidious nature of it, to the disease being confounded with abortion. The two diseases have been confounded so much in Ireland that it is quite a popular thing to think that an animal that is absolutely or relatively sterile is affected with contagious abortion. Perhaps the chief reason why this granular disease received very little attention is that if you examined a herd the owner would very likely tell you that it is the normal condition, if you find it; and if you go to any other herd in the vicinity you will have a large number showing these granules. Therefore, the thing was put down by many as a normal condition.

With regard to the lesions I have not found a typical acute case such as described by the German authorities. Granular lesions seem to be the primary lesions, and they are not preceded by any inflammatory stage characterized by discharge. As regards the contagiousness of this granular form, I will relate the experience I had, I think, about two months ago. I examined six virgin heifers and found five of them absolutely free from granular disease, but the sixth one had very slight nodules, something like swollen lymphatics—you could hardly call them granules. I examined the herd about six weeks afterwards, when five of these animals had been served by a bull, and all showed distinct granular lesions, while the sixth one had the vagina still healthy. That is one experience which leads me to think that the disease may be conveyed by coition. A point I should like to emphasize very much is that I have often found that the granular lesions are accompanied by an inflammatory condition of the mucous membrane, but with absolutely no discharge, or perhaps a very slight one. If you follow a case you will find the granular lesions fluctuate very much. A cow to-day may show these granular lesions in a distinctly swollen and red condition, and in three weeks or a month all the granules have disappeared, and you may have a normal condition of the mucous membrane; or very often you have a red condition with no discharge. That fluctuating condition of the lesions was another thing, perhaps, that may have caused the disease to be overlooked for a long time. Of 21 cases in one herd, six increased greatly after service, eight decreased from no apparent cause, and the remainder increased—quite a different feature as far as lesions are concerned in each group.

With regard to the acute disease I have not seen a typical case of it, but for these last two or three months I have been making post-mortems on the genital organs of cows at a slaughter-house in Dublin. In one case I came across what looked to me as showing, perhaps, an acute case. The vagina was covered with spots such as you would find in a bowel infected by acute swine fever: there was also some metritis, the womb containing a fairish quantity of yellowish pus. With regard to the cause of the lesions, Ostertag has put forward, and it has been confirmed by others, that the causes are streptococci. I examined a few scrapings from the vaginas of infected cattle, and I never could find streptococci. What I found were staphylococci and short bacilli. Another thing was that in cases of sterility I found that of sterile animals about five per cent. become infected with pyometra; I have had the opportunity of examining four of these cases, and in the pus-like matter I could find no organisms whatever, and any attempts at culture were negligible. With regard to bulls—speaking now of experience in herds where the sterility is fairly high and the vaginitis is widespread—the instance I have mentioned with regard to the six heifers goes some way to show that the bull carries the infection, but whether he conveys the disease mechanically or not I do not know. It appears from what we know of the disease on the Continent that he may be infected.

As to the treatment, I agree with the method laid down in the paper, but I must say that in badly infected herds I see no hope of getting rid of the disease by isolation. It would be most expensive where there are a large number of them infected. I have had some experience of the treatment by bacillol, according to Raebiger, giving capsules every four or five days, and then every fourth day for three or four days, and so on; and in very bad cases I have had them given every day for a considerable time, and the result has been up to the present no general or sustained improvement. The lesions fluctuate, and you never know where you are.

Dealing with sequelæ, I think there is absolutely no evidence to show whether abortion is in any way connected with vaginitis. I am sure that 80 or 90 per cent. of the cases of epizootic abortion, or perhaps more, are due to Bang's bacillus. The most important point we have to decide here to-day is the connection between vaginitis and sterility. You will find that most of the cases of epizootic abortion occur in the sixth or seventh month, or from the fourth month onward, of pregnancy.

I have had a good deal of experience of animals that would not conceive, or returning to the bull about the tenth or twelfth week. What I would like to know is: Is there any connection between vaginitis and that condition? For a long time I considered that there was, simply because I was dealing with herds where the sterility figure was high. I then went outside and found that I could get these granular lesions and fruitful animals in the same proportion almost as I could get them in the sterile herds. Although you find this granular form of vaginitis very often present, there is no distinct relation between it and sterility. There are other very interesting points in Mr. Wilson's paper with regard to the direct cause of sterility, such as ovarian and uterine diseases.

As a result of about 40 or 50 post mortem examinations I found a considerable number of lesions. The animals were a mixed lot, perfect maiden heifers up to old cows, and I found cystic degeneration of the ovaries rather a common thing, and also large corpus luteum. After that would come metritis, and I did not see a single case of vaginitis except the one I mentioned showing the spotting in the vagina. I should like to ask Mr. Wilson to describe exactly what he calls a cyst or enlarged corpus luteum, bucause I think a corpus luteum of a considerable size is a physiological condition. I should also like to know whether he will give us a rough outline of the technique of his operations on corpus luteum and cysts. I hope to-day, before we part, some resolutions will be passed and a committee appointed to go more fully into the matter.

Mr. A. RENFREW: Although it has really nothing to do with the paper, I might be permitted to ask such an eminent authority as Mr. Stockman whether I understood him to say that contagious abortion was as likely to take place at two or three months as at seven months.

Mr. STOCKMAN: It may, after infection.

Mr. RENFREW: I happen to be working in a very wide district amongst cattle, and I am sure that 90 per cent. of the cases of real contagious abortion take place at the seventh month, or two or three weeks afterwards, even when we know that contagion has been conveyed by a contagious bull.

Mr. STOCKMAN: That is not our experience. What I mean is that a very large number of the calves sent up to us are much earlier calves.

Mr. RENFREW: My point is that we get a cow that aborts at three or four months, and we hardly take any trouble to have the cow or the cleansing particularly cared for.

Mr. D. PUGH: I have had a little experience of this so-called granular disease in two herds. I was called to examine some cows which were supposed to be not fruitful; they were kept chiefly for show purposes. They had these granules in the vagina. I tested them with litmus paper, and three or four were distinctly acid in reaction, while the others were neutral. I gathered from the cowman that if the cows were served with one bull they never got in calf, although it was the best bull on the place, but that when they put a young bull on to these cows they become pregnant. It occurred to me to test the secretion from these bulls. The big bull was distinctly acid in the secretion from the sheath, but the discharge of the young bull was not. After this big bull had served the cow I passed the inseminator into the cow and collected some of the semen, put it into a test tube, and put it into a thermos flask. I got some also from the younger bull, and took it home in the same way. I examined them both under the microscope, and I found the vitality of the spermatozoa from the big bull was very weak ; they certainly wriggled about, but they did not seem to get forward in the semen. In the semen from the younger bull the spermatozoa were exceedingly active. I had all the cows washed out, and had the big bull attended to, and I told them not to allow the bull to serve the cows until the reaction from the sheath was normal. I gave them some blue litmus paper to test the bull. After a certain time the discharge became neutral, and they used the bull again, and he got the cows in calf. The second herd was also kept specially for show purposes, but the cows could not be got to calve at the particular time required. Even the heifers had these granules. There were two bulls, and I tested both, and both were acid, as were also two or three of the cows. I had the bulls thoroughly washed out, and there was no difficulty afterwards in getting these cows in calf.

My difficulty is to find cows without these granules, although there is no sterility in the herd. On page 69 Mr. Wilson says, "It is good practice to syringe out the vagina with a one per cent. solution of bi-carbonate of soda before going to the bull, in case acid or a neutral state of the mucus exists. The reaction should be feebly alkaline, and various conditions of the genital tract may alter this." Personally, I think that is not good practice. You are not going to affect that mucous membrane if it is acid with one washing before the bull covers the cow, and if you leave any fluid in the vagina to dilute the semen you reduce the vitality of the spermatozoa very considerably. You have only to put them under the microscope and drop a little alkaline solution on them to find the vitality is considerably reduced, and that you even kill a large number of them.

REPLY.

Mr. AINSWORTH WILSON : I should like to thank those who have taken part in the discussion very heartily, especially Sir John M'Fadyean for his criticisms. Really, one of my objects in writing this paper, and in writing it in its present form, was to provoke criticism. My difficulty was to find a herd in which the granules did not exist, and to find individual cows in that herd which were free of the disease or of the granular condition which I have characterised as a disease. Mr. Stockman has dealt much more kindly with me than I had anticipated. Apparently he has not done any extended work in the laboratory probably because he has not had the material, the disease, if present, not being very common in this country. Another of the objects for writing this paper was to determine whether the acute contagious disease actually existed. Sir John has found fault with me for calling the malady I have observed contagious granular vaginitis, but I have done nothing of the sort. I have described contagious granular vaginitis as it exists on the Continent, and I distinctly say in two or three portions of my paper that I have never seen the acute disease. Therefore, I am bound to go to foreign authors for a description of the acute form. But I still adhere to my opinion that the chronic lesions as described in the paper, are identical with those described by German and Swiss authors, absolutely identical in every way; and I have also distinctly referred to the fact that the chronic form is recognised as being non-contagious on the Continent. I have attempted to prove that the chronic form, as it exists in Essex, is non-contagious; therefore I am correct in concluding that my observations agree with those of continental authorities. As to whether the disease is preceded by an acute stage, I am, perhaps, wrong in describing it as the chronic form of the disease when there is no acute form. I have not observed an acute stage. It is said that you find it in calves at birth, but I have not seen it in calves at birth, though I have seen it in calves two or three months old. I have traced animals from birth upwards, and I have not found it preceded by any acute form. I have not noticed that it is infectious in any way, but that does not say to my mind that the disease is not a chronic or benign manifestation of the acute disease existing on the Continent. If the statement I have just made is bacteriologically incorrect, or pathological nonsense, I must ask your pardon, but that at least is the simple statement of affairs. I candidly admit that only twelve months ago I was unaware of the existence of this granular condition until Professor W. L. Williams, of New York Veterinary College, pointed it out to me. His account of the disease agrees with that of other authors, and coincides with what I have observed during the last twelve months. I think it is only right to call attention to the condition, in the event of the acute or contagious form manifesting itself at any time in this country. I was very interested to hear that both Sir John and Mr. Stockman have noticed the acute form, and I should like to ask them if they have ever published their observations? Mr. Stockman certainly says he has recognised the contagious disease in this country, and he cites two cases. He is my master in a bacteriological sense, and in most other respects, but I must differ from him here. He says with regard to acute gonorrhœal vaginitis, or acute vesicular vaginitis, that the granules were well developed in two cases. He describes a typical case of the vesicular disease in which the granules were present. I contend that it was quite possible—in fact we know—that these two diseases may co-exist, and I have given instances where the granular lesions were present in an extensive form before copulation, and after copulation the animal had the vesicular disease superimposed on the granular infection. There is no reason why they may not co-exist. In the human subject all three venereal diseases, or two of the three, may co-exist in the same person. If you admit that these granules are an abnormal condition in the vaginal mucous membrane, then we must regard them as a mild, benign form of disease, but as a disease, and they may co-exist with the vesicular disease—so-called cow clap, and I have noticed repeatedly that they may co exist. I fancy in these cases of Mr. Stockman's the granular affection was there beforehand, and the vesicular disease appeared afterwards as a result of copulation or infection in other ways; but I may be wrong. With regard to the etiology, I notice Mr. Stockman has failed to reproduce this disease with streptococci. The organism of Ostertag has not been generally accepted as the causal agent. Numerous writers have accepted this organism, but it is by no means clear they have actually gone in for original experiment. It seems to me that in many cases they have simply accepted the statements of others, and have not actually submitted it to proof. I had hoped to hear something to-day about these so-called Prowazek's bodies, which may or may not be protozoa. They have been found by a German, veterinary surgeon in acute cases of granular vaginitis, and also in trachoma of the human eye, and they may or may not be connected with the disease. It is interesting to note that granules occur on other mucous membranes. Mr. Henry Gray last year read a paper on Diseases of the Eye, before the Central Veterinary Society, and he said that in all dogs he had examined on the posterior surface of the conjunctiva there was a distinct granular condition, and he asked whether that was the normal condition or not? I ask to-day whether the non-inflammatory granular lesions in the vagina of cows and heifers is a normal condition? I am not prepared to say it is a disease. I call it a disease because it corresponds in every way with the chronic form of the disease on the Continent. I am rather going back on what I said before, but Dr. Hess and others recognise that the contagiousness of the disease disappears with the onset of the chronic stage. Mr. Stockman mentions that he has reproduced the vesicular disease with the thermometer, and one can well understand that; I have done the same thing myself.

With regard to abortion, I think one point should be cleared up. I do not really think Mr. Stockman meant to say that the majority of cases occur in the fourth month, or in the earlier stages of pregnancy. I think he will agree with me that the majority of cases of epizootic abortion occur somewhere about the sixth, or between the sixth and the seventh month. In the three hundred cases I have cited, of which a careful record was kept, the average number of cases occurred between six and six and a half months. That does not mean to say that cases did not occur at two months. I have two months' old fœtuses in pickle from herds in which contagious abortion was very abundant, and I have had them at all ages. At the same time I think it will be found that somewhere between six and six and a half is the more usual time.

Mr. STOCKMAN : Do you disregard the abortions at four months?

Mr. AINSWORTH WILSON : Not at all. Every abortion is reckoned from the time it is observed. No doubt some cases were not observed in the earlier stages—in fact, I think unnoticed or "invisible" abortion is rather common in some herds, and accounts for some cows supposed to be in calf and coming in œstrum again after some months ; they have slipped a very small fœtus which has never been noticed.

Mr. NORRIS : Is that contagious abortion ?

Mr. WILSON : If you get it in a herd where contagious abortion is very common, and where a great number of cows keep on slipping, you are entitled to so regard it.

Mr. NORRIS : Have you examined any of the discharge ?

Mr. WILSON : Not in the early cases, because it is generally contaminated. The cases I have examined have been those sent to isolation farms usually further advanced in pregnancy. The observation has been made that sterility is certainly prevalent in animals free from vaginitis, and Mr. Stockman criticises Hess on this point. I think he is certainly right, and my remarks are in the same strain.

Sir John M'Fadyean condoles with me, but I must confess I do not feel very down in the mouth with his remarks ; in fact, I take them in the right spirit. He is quite right, of course, to say that the disease which I have described is not contagious granular vaginitis. I never pretended it was. As I said before, I have described the contagious disease from foreign writings, and then I have described a form which is prevalent in my county, and which appears to be identical with the chronic form abroad, and in neither of which is contagion a factor. My description does not conflict at all with that of German writers, except that I have ventured to say that they have attached rather too much importance to the disease abroad, and that they have ascribed almost every abortion, and in some herds practically all the sterility to this disease. I think I am justified in saying that they have overstepped the mark, because infectious diseases of the genital organs are only one of the factors in connection with sterility, which, as we all know, is due to a great variety of causes. Professor Hess recognises this. I do not deny that contagious granular vaginitis may cause abortion. I have only tried to show that the chronic, or mild benign form is not associated with abortion in my practice. Sir John asked me if it was not the case that on the continent it is accepted that where the contagious disease exists 50 per cent of the animals are sterile.

Sir JOHN M'FADYEAN: 20 to 50 per cent.

Mr. AINSWORTH WILSON: Undoubtedly. The percentage is given as 10 to 70 per cent. by various writers. None of us in this room are in a position to deny this, simply because we have no experience of the acute disease—at least no one except Sir John himself, who has seen something of it, at any rate. I think he rather found fault with me because I dared to describe this disease when I had not seen a case. I have described the mild granular lesions which alone manifested themselves, and for the acute form I have had to go elsewhere, simply because I could not discover an acute case.

Sir JOHN M'FADYEAN: I have been wondering what is the explanation of the disease spreading until it involves 80 per cent. in the chronic form. If it is non-contagious and you have never seen an acute case, how do you account for the disease spreading until 80 per cent. are affected with the chronic form?

Mr. WILSON: I admit that the point raised is a very important one, and to a certain extent I anticipated it. What I meant by saying the chronic form is not contagious is that it is not contagious in the ordinary way; it is not contagious by handling or by coition, so far as I have observed, but at the same time the percentage of animals affected does increase remarkably as age advances, and I am not prepared to say how it is that the lesions advance with age, and I am not prepared to say why so many animals are affected. I am seeking for information, like everybody else. But I am prepared to say that the chronic form on the Continent is exactly like that which I can show you any day, or which you can see for yourselves. Mr. Norris certainly has had a somewhat remarkable experience in herds where the sterility figure was over 50 per cent. I have seen some pretty bad herds, but the sterility figure was never over 20 per cent. One observation made by Mr. Norris was rather important, although it does not prove anything. He spoke of six virgin heifers, one of which showed slight lesions, the other five were free. On a subsequent visit, after five of them had been to the bull, they all showed granular lesions. I cannot account for that in any way, unless the bull was affected prior to serving these heifers. I do not know whether he mentioned the length of time that elapsed between his visits.

Mr. NORRIS: Five or six weeks.

Mr. WILSON: All I can say is that I have never observed it, and I have watched them, particularly after copulation. You do get an irritable condition after copulation in an animal in which the granular lesions are extensive. Mr. Renfrew remarked with regard to epizootic abortion that seven months was a common period in his experience. I suppose he strikes an average in all his cases. Mr. Pugh's remarks with regard to the reaction of the secretion are interesting. He found fault with the vaginal injection of one per cent. of carbonate of soda. I have injected mares immediately before service, and also cows the day before and immediately before copulation, without in any way retarding conception—in fact, aiding it where the reaction has been acid. In some cases of sterility where the examination had not been made with litmus paper or otherwise, I recommend the injections, only, unless I do it myself, I generally advise them to do it the day before. There is no reason, however, why you should not do it immediately before, so far as my experience goes. In conclusion, I recommended that a Committee should be formed to ascertain whether contagious granular vaginitis actually exists in this country. If it does exist, it may be, and probably is, an important factor in those herds in which the sterility figure is very high and remains high.

STRANGLES.

Capt. A. G. Todd, A.V.C.

Strangles was one of the first of equine diseases described by the earliest writers in veterinary science. They remarked on its enzootic character, its contagiousness, and its frequency in young animals compared with old. Solleysel described it, in 1664, as a disease which young horses had to pass through in the same way as children had to pass through small-pox. Convinced of its contagious nature, he recommended the isolation of the affected, and pointed out that the most common way for horses to become infected was by drinking out of buckets which had been previously used for affected animals. Lafosse proved by experiments, in 1790, that it was contagious, and Viborg confirmed this in 1802.

In 1873 Rivolta observed chains of cocci in the pus of abscesses, but Schutz (9) in 1888, was the first to publish a detailed description of the organism. Sand, Jensen, Poels, Baruchello and many others have since contributed to the bacteriology of the subject.

The cause of strangles is the streptococcus equi, which is found in large numbers in the nasal discharge and abscesses, which are characteristic of the disease. In unbroken abscesses it is found pure, but in the nasal discharge it is associated with other microbes. The prevailing form of organism is an undulating chain of cocci, each chain being made up of 30 to 60 organisms. The length of the chain varies with the situation ; in throat abscesses they are longer than in the nasal discharge, where individual cocci, diplococci and short chains are principally found. This variation in the length of the chains is due to its surroundings. A similar condition may be observed in different culture media, the more liquid they are the longer the chain, and *vice versa*. The method of reproduction is by transverse division.

The organism stains well with all aniline dyes, and retains the stain by the method of Gram.

BIOLOGICAL CHARACTERS

Growth takes place on most media at room temperature. It is both ærobic and anærobic, and is non-motile.

Bouillon.—In bouillon a white deposit may be seen after 24 hours growth resting at the bottom of the tube. When shaken the fluid becomes turbid, but the deposit soon settles leaving the upper part of the tube clear. The growth, which consists mostly of long chains of streptococci, is more abundant when serum is added to the medium.

Blood Serum.—On the surface of blood serum typical, sticky, transparent, colourless droplets form which are separate at first, but afterwards confluent. In the water of condensation which collects in the bottom of the tube, a sediment forms which consists of long chains of streptococci.

Agar.—On agar slants colonies appear which are in the form of small, flat, circular, greyish white spots ; these are never confluent and are easily overlooked.

Gelatine.—On gelatine it grows as very small white spots, but the growth is never abundant, owing to the low temperature at which gelatine has to be incubated.

Potato.—There is no visible growth on potato. Sand and Jensen state that it grows into the surface of the potato and cannot be seen, but I have not been able to verify this.

Milk.—It grows in milk, causing curdling in six to eight days.

Glucose.—There is no gas evolution.

Vitality.—A temperature of 60 C kills it in one hour.
,, 80 C ,, half ,,
,, 100 C ,, a few minutes.
Sunlight destroys it in eight hours, and dessication in three weeks. (1) It is killed in 15 minutes by the following :—Perchloride of mercury 1-1500, Carbolic Acid 1-200, Creolin 1-80. (2).

Cold does not appear to affect it as much as heat, bouillon cultures frozen for two days are still capable of growing when brought back to a suitable temperature. Although it is easily cultivated in the various laboratory media, its virulence decreases even under the most favourable conditions of artificial cultivation. To bring it back to its original virulence it is necessary to pass it through some susceptible animal.

PATHOGENESIS.

White mice die in two to four days when inoculated subcutaneously with minute doses of pus or cultures. The infection is general and the organism can be isolated from the liver, spleen, kidneys, peritoneal cavity, lungs, heart, blood and lymph glands. There is abscess formation at the seat of inoculation, and if death is delayed, as is the case when organisms of low virulence are used, abscesses form in adjacent lymphatics, and afterwards in the lungs, liver, kidneys and other organs.

Rabbits and guinea pigs are not very susceptible to the streptococcus. Large doses injected into the veins or peritoneal cavity are necessary to kill them. Birds, dogs, swine, sheep and cattle do not contract the disease when inoculated with cultures.

Local inflammation, necrosis and abscess formation results when horses are inoculated subcutaneously, and this may or may not be accompanied by systemic disturbance. Intravenously small doses cause a thermic reaction lasting 24 to 30 hours and no general symptoms. Strangles pus rubbed into the skin of healthy horses is said by Joly to produce a typical eruption, but in the few observations I have made on horses of five years of age and over I have not been able to produce any eruption, even when the skin was scarified.

Strangles pus rubbed into the nasal mucous membrane, or coming in contact with any mucous membrane, is often stated to be a sure way of transmitting the disease, but the following observations tends to show that, at any rate in old animals, additional influences are necessary. The nostrils of three horses, aged 6, 11, and 14 respectively, were smeared with both strangles pus and virulent culture, (pus in one nostril and culture in the other) and they were kept under observation for one month; the results were negative.

Another horse, aged 14 years, had 20 c.c. of virulent pus placed at the back of his mouth, and he remained in perfect health for two months afterwards, when he was destroyed for incurable lameness. Post-mortem examination revealed nothing abnormal as a result of the observation. Three other horses under orders for destruction, aged 8, 11, and 15 respectively, were given gelatine capsule balls, each containing 10 c.c. pus from a fresh strangles abscess; their temperatures were afterwards taken twice daily, and they were kept under close observation for a fortnight, during which time they were in perfect health. They were then destroyed and a careful post-mortem examination made, but not the slightest abnormality could be found.

These observations would undoubtedly be of more value if they had been made on foals, but they are interesting in that they show that aged horses in good health and living under favourable conditions possess an extraordinary high degree of resistance to enormous quantities of virulent infective material. The same pus inoculated subcutaneously into mice caused death in four days.

Schutz, Sand, and Jensen say that the disease is more likely to develop when, before the organism is introduced, the nasal mucous membrane is injured or in a state of catarrh. In one old horse placed at my disposal, a virulent culture rubbed into an abraded Schneiderian membrane did not produce the slightest effect. In catarrh, in addition to a large mucous surface being denuded of its protective covering, an animal's vitality is lowered considerably, so that one can understand such animals being more susceptible than others, but I have not had an opportunity of testing the susceptibility in such cases. No matter at what part the streptococcus enters the body, it does not ordinarily extend far from the point of entrance. For this reason the upper air passages and throat are most frequently affected. Its extension is principally along the lines of the lymphatic circulation. It may be carried by the blood stream to distant parts of the body, but this is fortunately rare, otherwise malignant strangles would be more common than it now is.

Although it is within the bounds of possibility for the streptococcus alone to cause the disease to develop in susceptible animals, clinical and experimental evidence point to an additional factor being necesary in the majority of cases. Some observers have suggested that the way is paved for the streptococcus by an invisible virus, and others have stated it requires the aid of other organisms, but no observations of any value have been brought forward to support these views. It is certain, however, that predisposition plays an important part in the development of the disease.

Age is a well established predisposing cause. It can be safely said that predisposition is inversely proportionate to the age. Viborg and Hertwig (2) observed that 70 per cent. of cases occur in horses under five years of age, 20 per cent. over five years of age, and 10 per cent. in horses over fifteen years of age.

Adverse climatic conditions, long train journeys, domestication, fatigue, standing about at fairs, change of food, changes of stabling, recent illness, in fact anything which produces a temporary lowering of vitality increases susceptibility.

An outbreak of the disease is described in Champetier and Payrou's "Etude sur la Gourme" (6) which well illustrates the influence of hardships, cold and fatigue. During the strikes at Limoges, in France, several brigades of mounted police were sent from distant parts to assist in quelling the disturbances and keeping order. Some of their horses were under five years of age, but the majority were over. Nights were cold, work long and severe, and the feeding very irregular. To protect them as much as possible from the inclemency of the weather, they were picketted in the riding schools of the cavalry regiments of the district. Strangles broke out simultaneously and indiscriminately in old and young; nearly every horse was attacked. During their work they were in frequent contact with the horses of the local cavalry regiments; intercommunication between them was unavoidable, but the disease did not extend to the cavalry. Moreover, when the mounted police left Limoges at the termination of the strikes, there was no time to carry out disinfection of the buildings before they were re-occupied by the cavalry, and although the cavalry horses were of all ages, young as well as old, not one case of strangles was observed.

Vitality in young animals fluctuates much more than it does in old ones, which perhaps accounts for age being such a prominent predisposing cause. Sudden changes of any kind, even though they may be for the better, at first produce a temporary loss of resisting power, which predisposes them to any prevailing disease.

Picquet (3) has seen remounts suffer from strangles after purchase in Hamburgh, again after recovery when moved inland to Hanover, and again after they have joined their regiment on the frontier. This, although rare, is a good instance of naturally acquired immunity failing to protect when sudden changes are made in the animals' surroundings, and should not be forgotten when artificially immunising horses.

In discussing the vitality of the organism, and its biological characters, it was pointed out that it could resist dessication for three weeks, so that in tracing the origin of an outbreak one has to consider the danger from infected forage, bedding, manure, water troughs, mangers, or any part of the stable contaminated by nasal or abscess discharges. It is quite possible for any of these to be the infecting medium, but if we take into consideration the rapidity with which it loses its virulence, even under the most favourable laboratory conditions, it is not unreasonable to conclude that the danger from any of these sources would depend on the length of time the organism had left the body of an affected animal. This is supported by laboratory observations. It is only with the greatest difficulty that one can get exposed infective material to grow on the most favourite medium. Strains of the organism whose virulence is on the decline can, however, easily be fortified by passing them through susceptible animals.

In remount stables and on board ship the source of the organism is nearly always an animal in the incubative stage of the disease. At

Lusk remount depot, from April 1st, 1906, to March 31st, 1907, out of a total of 33 cases of strangles, 15 were so affected on arrival, and in the following year, during the same period 11 out of 43 were similarly affected on arrival, But it is not in such outbreaks as these that there is any difficulty in tracing the origin.

Strangles occasionally breaks out in stables which have had no recent arrivals and where there has been no known contact with infected horses. An example of this is where remounts have finished their training, are transferred to the squadron stables, and one or two of them will develop the disease. Although one feels convinced that the source of the infection has not been outside the stable, a careful examination of all the horses will not reveal even a suspicion of a case of strangles. How are such cases to be accounted for?

There are some who maintain that the Streptococcus equi is a normal parasite of the upper air passages of equines, and that it only becomes pathogenic under certain conditions. This has not been supported by any scientific observations, and has therefore no real value. What appear to be responsible for this hypothesis is that a streptoccocus is found in the upper air passages of healthy horses which is indistinguishable from the Streptococcus equi. Future research may demonstrate that there is a difference between them, but it does not alter the fact that the Streptococcus equi can and does remain in the glands and follicles of the nasal mucous membrane of a certain percentage of recovered horses for a considerable time after recovery. They are harmless to such animals, because of the immunity conferred by the recent attack, and it is highly probable that some of these retain the streptococcus indefinitely and become "carriers" of the disease.

Infection.

There are three ways to be considered by which an animal may become infected:—1 By inhalation: 2, by direct contact: 3, by ingestion.

As the upper air passages are more often affected than any other part of the body it might be thought that inhalation would be one of the principal channels of infection. It would necessitate the organism entering the body in a dessicated state. Recent research has shown that this method of infection in other diseases of the air passages is very uncertain, and as the Streptococcus equi is no exception to the influence of dessication one may safely conclude that, although possible, it is unusual for the disease to be transmitted in this way.

Mammary strangles transmitted to the dam by a sucking foal is a good example of transmission by actual contact, as is also genital strangles of the mare transmitted by an infected stallion trying a mare.

There is abundant clinical evidence in favour of ingestion being by far the most common method of infection, and the irregular way in which the cases occur in an outbreak point to the watering trough as being the intermediate channel of infection. In the best civil stables, and in those remount depots where every horse has his own bucket, it is hardly ever seen. A striking example of this is the remount depot at Melton Mowbray where, out of 263 horses which joined from April 1, 1908, to March 31, 1909, only two developed the disease. One of these developed it within eight days of joining, the probability being that it had it in the incubative stage on arrival. From April 1, 1909, to March 31, 1910, out of 225 horses which joined there was not even one case of strangles. Although many of these horses were six years old or over, one-third of the number were four or five years of age.

Most horses when drinking bury the mouth and nostrils in the water, so that small particles of nasal discharge which escape ordinary observation are easily deposited in the water. In examining water troughs at which horses in apparent health have watered, flakes of mucus may be found which on microscopical examination will reveal streptococci and other organisms, and although I have not yet been able to establish the identity of all of them, there is no doubt about the presence of the Streptococcus equi.

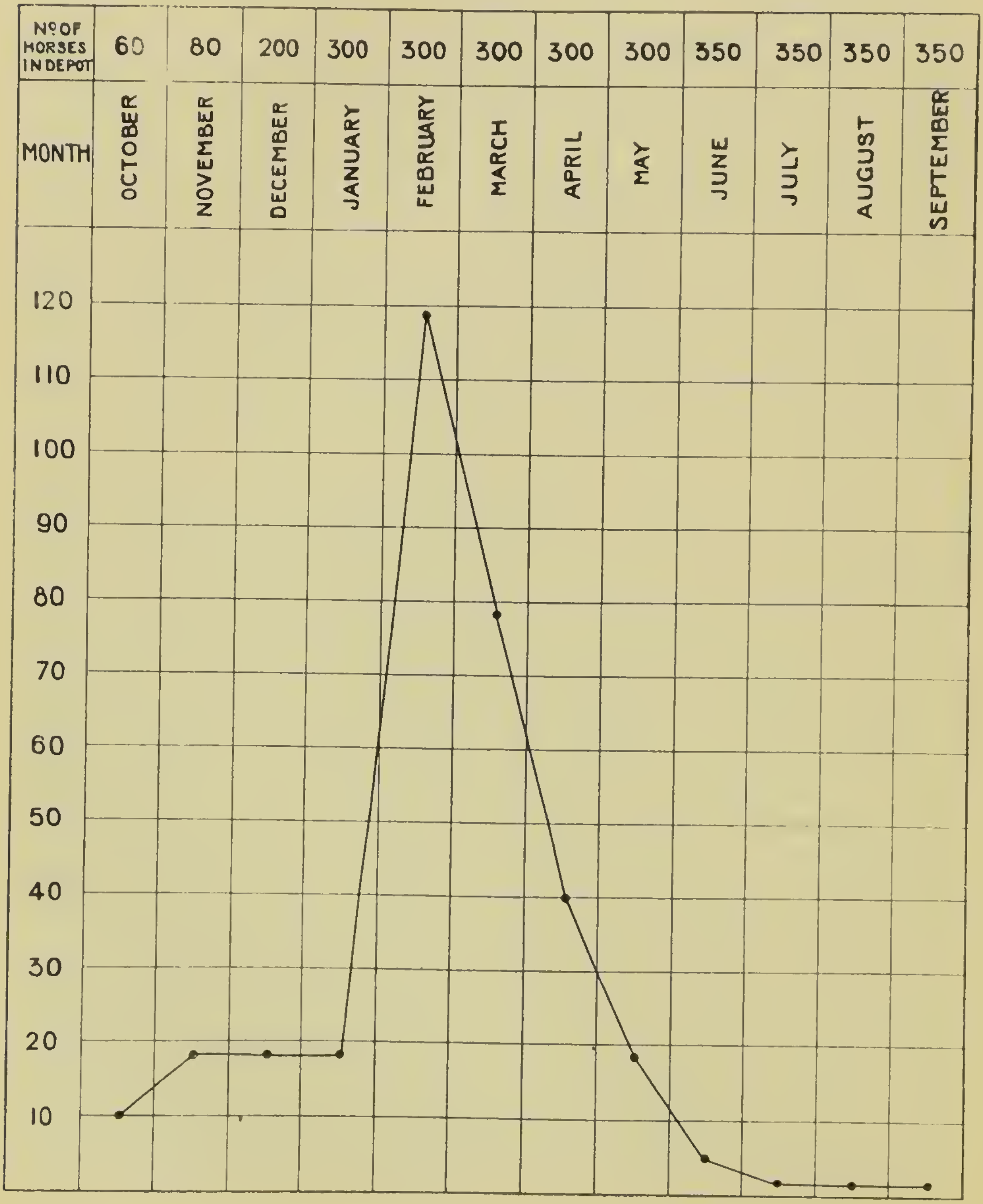
When large numbers of young horses are collected together, as in fairs, dealers stables, and railway trucks, it is impossible to know how recently some of them may have recovered from the disease, and watering as they do out of the same buckets or troughs, it is an easy matter for infection to be spread amongst them.

The simple form of the disease, viz., that associated with nasal discharge, sore throat, and submaxillary or parotid abscess, is that which is most commonly seen in England. The malignant form is seen mostly where hygienic conditions are bad, and in establishments where large numbers of young horses are kept together. We are more fortunate than our military colleagues on the Continent in this respect, in having a much milder form of the disease to deal with. Continental horse management is different to ours and may, in some degree, contribute to this.

In a continental Army with which I am familiar the remounts are bought at three and four years of age, and remain at the depots until they are five years of age, when they are issued to regiments. This necessitates the keeping of some 300 or 350 horses together for about a year. It affords unique opportunities for making observations on the course of an outbreak, reference to which is both interesting and instructive, especially if at some future time we adopt a similar system in England.

In the Army to which I refer remounts are bought at fairs or at previously arranged centres in breeding districts; purchasing begins in October and goes on till June. After purchase they are sent by train to the depots, where they are turned out to graze. The first part of the procedure entails irregular watering and feeding, possibly a railway journey to the fair and another to the remount depot, and a good deal of standing about, all of which predispose to the disease. The result is, that some arrive at the depots clinically affected, and many of the others develop the disease shortly after arrival.

The largest numbers are bought during December, January, and February, so that batches are continually arriving. Atmospheric variations at this time are frequent, and the horses feel the effects of these journeys and changes more than at other times. Grazing is scarce, which necessitates feeding the horses in sheds, where they huddle together for protection against wind and rain. Cases now become numerous, and it is at this time that malignant forms of the disease are mostly seen. With the improvement of the weather and the increase in the grazing, the horses leave the sheds for the open fields, with the result that there is an enormous drop in the percentage of sick, and by the beginning of summer the outbreak is practically over. This is the usual evolution of the disease at the continental remount depot I have in my mind, but there are occasional years, more especially when the winter has been a very mild one, when the disease is at its height as late as May or June. The following chart shows a rise and fall in the number of cases, from the time they come to the time they leave the depot. It illustrates the influence of unfavourable weather, and the evils of crowding large numbers of susceptible animals together at such critical times.



The above system is conducive to most horses passing through the disease before their issue to regiments, but whether it is an advantage or not is doubtful. The high percentage of malignant cases, the mortality, and the number which are left unsound in the wind after the disease are not in its favour.

The mortality varies from 0 to 3 per cent. When hygenic conditions are bad it is high. The deaths are mostly from malignant strangles, and more amongst young than old. On the Hungarian State Stud Farms, from 1886 to 1889 1711 foals, almost exclusively under one year, were attacked, of which 48 died or 2·8 per cent. At the same time the disease was prevalent in the State Stallion depot, and out of 118 stallions attacked there was only one case of malignant strangles which was destroyed.

In the Prussian cavalry the mortality varies in different years from 0 to 3·4 per cent. From 1900 to 1903, out of 3,954 remounts affected 82 or 2·1 per cent. died. In the Prussian remount depot from 1899 to 1906 out of 1,328 horses attacked 35 or 2·6 per cent. died.

In the French Army from 1888 to 1897, out of 90,381 horses attacked 992 or 1·1 per cent. died. From 1900 to 1901, out of an establishment of 100,000 horses there were 18,165 cases and 283 (1·5 per cent.) deaths.

In the English Army over a period of years the average death rate was 0·76 per cent.

SYMPTOMS: INCIDENCE.

The symptoms of strangles are so well known that there is no need to describe them here. There are one or two points, however, to which I propose to refer, as they will have to be considered in dealing with the prevention of the disease. There are two forms of the disease recognised in England, viz., simple and malignant strangles. To these should be added atypical strangles; it would include these forms of catarrh, sore throat, and catarrhal fever which are caused by the streptococcus equi. Until recent years these diseases have been looked upon as being quite distinct from strangles, but, although laboratory methods and clinical observations have as yet failed to provide a distinguishing difference between the typical and atypical forms, there is abundant evidence to prove that the latter are often the beginning of the disease. Percivall (5) observed this, and in a paper read before the Central Veterinary Society last year W. R. Davis (7) gave one or two good instances of catarrhal affections in old horses being the origin of the typical forms of the disease in young ones. Horses over six years of age and those having secondary attacks of the disease usually have it in the atypical form. It is best to consider any form of catarrh in a remount stable as specific, and more especially when some of the others have the disease in the ordinary form.

Out of 930 remounts passing through Lusk remount depot from 1906 to 1909, 82, (0·88 per cent.) had typical strangles, and 39 (0·41 per cent.) had symptoms of catarrh only; although the latter had to be entered on the sick returns as catarrh it is extremely probable, that some of them were mild cases of strangles. It may be said with almost positive certainty that long-continued catarrh in young horses, especially the form known as catarrhal fever, is suppressed strangles; suppressed in the sense that there is no external abscess, but the pharynx of such a case has abscesses in it which are the source of the discharge.

A natural attack of the disease confers a varying amount of immunity, the duration of which is difficult to ascertain. Humbert (2) records a four years experience amongst horses over two years of age in which 2195 had it once, 543 twice, and 121 three times, and 1641 remained unaffected. It is not stated whether atypical forms of the disease are included, and as secondary attacks sometimes assume these forms, these figures may give an erroneous idea of the recurrence of the disease.

Preventive Measures.

Owing to the facility with which horses recover from strangles, and the slow and uncertain manner in which it spreads in the ordinary way, preventive measures are rarely adopted. The breeder does not look upon it seriously because very few die from it, and most animals on farms recover with no after effects. The sufferers are these who keep large numbers of young horses together, at the time when they are first put into work. Here the loss is more from depreciation of value from unsoundness in the wind following on the disease than from actual mortality. Serious as this loss is, it is nearly always accepted as inevitable.

In making an organised effort to keep away the disease and to prevent its spread to others, the following measures should be adopted. All new arrivals should be quarantined for a fortnight before being allowed to join the other horses. The period of incubation being from three to eight days this would allow ample time for the disease to develop if they had it in the latent form.

Where only a few horses are kept this is a simple matter, but where horses arrive in fairly large numbers, as they do when joining regiments, it is not so easy, and special precautions have to be taken. The stables for their reception should be so constructed as to make it impossible for one horse to dip his nose into the manger of his neighbour or touch any of his neighbour's food. This can be effected by having a stall partition or by the ring and sliding bar method of tying up, the collar chain never being more than 2ft. 9in. in length.

The bedding of all horses should, after the soiled parts are removed, be allowed to remain in the stalls, especially for the first fortnight. The system of taking it out of the stalls and allowing it to mix with the general supply of the stable is apt to spread other diseases as well as strangles. With strangles the danger is more from eating infected bedding than from skin contact with it. For this reason peat moss would be preferable to straw.

As the bucket system of watering in regiments is out of the question on the score of expense, and the risk there would be of buckets getting mixed, a common watering trough can be used, provided it is emptied and washed out after every watering parade ; this would ensure that no unobservable discharge was left in the water.

Before each watering parade there should be a nose and throat inspection by the N.C.O. in charge, and an officer's inspection at least once a day. Any horse with the slightest suspicion of nasal discharge should be prevented from watering at the common drinking trough, and reported to the veterinary officer in charge. Major F. Eassie, D.S.O., A.V.C., has devised a system, which is now adopted at Lusk remount depot, by which all remounts out at grass go through a "crush" once daily without the trouble of being caught up. They receive a feed of corn once in the day, previous to which a bell is rung. This is the signal for them to come from all parts of the field and walk through the "crush" into a feeding paddock. A well trained N.C.O. stands at the end of the "crush," and secures and isolates any horse with nasal discharge. By making this daily inspection strangles has been kept well under control at Lusk.

Particular attention should be paid to the ventilation of the stable. Young horses cannot have too much fresh air. In addition to modifying most of the diseases they suffer from, it often keeps them robust enough to escape infection.

When cases have been diagnosed the stall should be evacuated and thoroughly disinfected, particular attention being paid to those parts which have been soiled by nasal or abscess discharge.

The bedding should be burnt as well as any food which may have been left in the manger. In remount stables the disinfection of the whole stable is unnecessary in isolated cases, but when the disease has spread through most of the occupants it should be thoroughly disinfected before re-occupation. This can be thoroughly and effectually done by using a blow lamp on the walls, floors, and mangers, and afterwards, applying a suitable disinfectant with a spray pump.

IMMUNIZATION.

Artificial attempts at producing immunity have been many and varied. The Russian method is the oldest and at the same time the crudest. It consists in turning the animals into a deep pool for half an hour, exposing them to cold winds, and giving them very cold water to drink. This gives the organism every chance of gaining access to the system, and the result is that the animals are got through the disease in the mild autumn weather and sufficient immunity is conferred to carry them through the very severe Russian winter, during which mortality is very high in the unprotected.

Jensen and Sand injected cultures intravenously, and found that, although they conferred protection against artificial nasal infection, they were dangerous on account of the phlebitis and local inflammation which they frequently set up.

Kitt, in 1906, injected two foals with 5 to 10 c.c. of killed serum bouillon culture and found that they afterwards resisted nasal and bowel infection.

Gabritchewski injected subcutaneously concentrated bouillon cultures evaporated down to one-tenth their volume and sterilised with 0·5 per cent. carbolic acid. Six foals were inoculated and they afterwards withstood virulent cultures being rubbed into the nasal mucous membrane. They also showed higher resistance than the controls to 1·5 to 2 c.c. of virulent culture subcutaneously.

Baldrey (10) working on the lines laid down by Wright, vaccinated rabbits and found that they afterwards resisted subcutaneous inoculation of virulent cultures.

No practical method of immunization suitable for preventing the disease in large studs, has yet been devised from either living or dead organisms. More encouraging results have been obtained from the serum of recovered or hyper-immunised horses.

Delvos used the serum of recovered horses for protection in 20 c.c. doses subcutaneously, and found that 94 horses so inoculated did not contract the disease and that sick animals receiving 30 to 40 c.c. repeatedly recovered in a very short time.

Rohrs found that with serum injection in the treatment of the disease there was less nasal discharge, not so much fever, and resorption of abscesses on the point of development. He employed it in 10 c.c. doses three times a day on the first, second, and third days.

Jacoulet noticed that fever was of shorter duration, complications and relapses less frequent, and that the patients got better sooner, when treated with serum. A daily dose of 20 to 30 c.c. was given; 80 to 100 c.c. brought the disease to a standstill.

Dassonville and Wissocq (8) have shown that by injecting horses with progressively increasing doses of strangles streptococci a protective serum can be produced, which confers a passive immunity lasting from four to six weeks. Their method of hyper-immunization consists of giving 2 c.c. doses of cultures subcutaneously and gradually working up to 100 c.c. doses intravenously. Twenty-five or more injections are necessary before the maximum dose can be given. They tried to convert the passive immunity into a permanent one by injecting cultures of the organism about the same time as the serum, but had to abandon the method on account of the severity of the local lesions. The French Army tried the Dassonville serum but did not adopt it for general use. Civil Veterinary Surgeons in and about Paris appear to have had remarkably good results from the serum.

Desoubry (12) at a recent meeting of the Societé Centrale de Médecine Vétérinaire, records having successfully inoculated 650 animals between 1st April, 1907, and 31st May, 1909, at places where the disease ordinarily occurred; M. Desoubry's practice is mostly amongst thoroughbreds, and his plan is to give a 20 c.c. dose to all foals *(a)* at birth or shortly after, *(b)* at the time of weaning, *(c)* when they are sent to their training establishments, *(d)* when an outbreak occurs in a stud, to immunize those exposed to infection.

Poels, of Rotterdam, produces a serum by hyper-immunizing horses with weekly injections of virulent cultures, beginning with 1 c.c. and increasing the dose gradually up to 20 c.c. After a total of 100 c.c. has been injected the serum is rich enough in protective substances to be used for immunizing horses. The dose of the serum is 10 c.c.

Jess, Piorkowski, Hoecht and Gantz, in Germany, have also produced protective sera which have found favour with some and not with others.

The reason why serum inoculation against the disease has not been generally adopted is partly because of the high price of the serum and partly because the immunity produced is not a permanent one. There are occasions, however, when a temporary immunity can be utilised to great advantage, and although it has not as yet found much favour in England it has been put to good practical use in other countries.

The duration of the immunity conferred by serum is not exactly known. With most other diseases it begins to decline after the tenth day and has disappeared by the end of the third week. It has been pointed out by Basset (13) that in the case of a homologous serum, viz., that used on an animal of the same species as that from which it has been prepared, the immunity is twice as long as that conferred by a heterologous serum, viz., that prepared from an animal of a different species. In the first case it is said to be three or four weeks and in the second about twelve days.

The size of the dose also, has, no doubt, some relation to the duration of the immunity. In France a larger initial dose is injected in preference to repeated small doses. Their reason for doing so is, that recent researches have shown that when anti-bodies, which are the protective agents in anti-sera, are injected into animals, they provoke the formation of corresponding anti-bodies. Secondary doses are neutralised more quickly than the first, so that there is a smaller production of anti-bodies and a corresponding shorter period of immunity conferred.

For most practical purposes in the Army one initial dose of 20 c.c. would perhaps be better than repeated small doses, but this is a point which only practical experience can decide.

In the service the time that remounts most require protection is (1) when they are bought, (2) on transfer from a remount depot to a unit, (3) on transfer from the remount stable to a squadron stable. On all these occasions there is a lowering of vitality and a temporary breakdown of an animal's resistive power, which, providing the streptococcus is about, is conducive to the development of the disease.

I regret that I am not in a position to give exact statistics on the preventive value of serum in the English Army, but I am sure I am safe in saying that the immunity conferred may be compared with that acquired by an attack of the disease. It may not be quite so marked, but it is better in that the disease often leaves after effects, in addition to immunity, which seriously depreciate an animal's value. Secondary attacks in unprotected horses are mostly in the atypical form of the disease, and it has been observed that when serumized horses do get the disease, it is usually in this or a correspondingly mild form. Further, the dreaded after effects are seldom seen in serumized horses. Of 69 protected remounts in the Aldershot command, only one was found to be unsound in the wind when examined after convalescence. This one had never shown any symptoms of strangles or catarrh, so it is reasonable to conclude that it was due to some other cause. Compared with the above, 9 out of 18 unprotected remounts were in various stages of wind unsoundness. This is a very high percentage, as ordinarily only 20 to 25 per cent. are left unsound in the wind, but even this is far too high, and anything which reduces it warrants its use till something is discovered which will give permanent and absolute protection against the disease.

We have not had the excellent curative results which some of our continental *confrères* have experienced, but in many cases treated, serum seemed to modify the attack and the progress of the disease; animals became brighter, the character of the nasal and abscess discharge changed, and in some instances throat swellings, which in ordinary cases we would expect to develop into abscesses, gradually subsided without abscess formation.

It does not follow that because horses have been inoculated against the disease, disinfection and other hygienic rules should not be observed. It is only by using a combination of both that any real benefit will result.

Palliative Treatment.

Strangles being a specific disease and running a definite course, treatment is essentially palliative. Too much stress cannot be laid on a good supply of pure air. It is well known that animals turned out to grass seldom get the disease in a severe form, and usually recover without complications or after effects, whereas the reverse is the case when animals are kept in large numbers indoors. In the service the animal is usually put in a box in the hospital, but sometimes, owing to the large number affected, they have to be kept in stalls. Personally I would prefer to see them picketed in the open at any time of the year, if boxes were not available. Recovery would be much quicker, and complications and after effects much more rare if this was adopted.

Food should be of the laxative type, and easily digestive. There is nothing better than an abundance of green food when it is in season, which, in addition to being laxative, cooling, and easily digested, assists in eliminating the toxins from the system by reason of its laxative and diuretic properties. All food should for preference be given off the ground to allow nasal discharge to drain freely away, and it is better to put it in small quantities round the box than in one place, so as to avoid its being contaminated by the discharge. When the threat is very sore, the food sometimes collects and ferments in the back part of the mouth and pharynx through the horse being afraid to swallow. In all cases when dysphagia is pronounced it is better, for this reason, to withold all solid food, and to rely on gruel, milk, linseed tea, and even water till the symptoms are relieved.

Water is best given in separate buckets, and frequently changed, as it soon gets filthy from the nasal discharge. When the throat is very sore it should have the chill taken off to prevent paroxysms of coughing coming on. For all ordinary purposes, however, cold water is best.

Steaming gives relief when there are internal throat abscesses and when there is excessive nasal discharge. Washing the nostrils and smearing them with vaseline facilitates the flow of the discharge, and prevents it from collecting around the nostrils, Details such as these would come under the heading of good nursing, and are very essential in all severe forms of the disease.

Medicinally very little is required. Salts of potash in the drinking water and electuaries to relieve soreness of the throat are the most frequent remedies used. Clive Webb (11), found citric acid in one drachm doses dissolved in water, three times daily for three days, of the greatest value in relieving dyspnœa, and attributes its success to its action on the coagulability of the blood. It encouraged the transudation of lymph and thereby aids bacterial defence; at the same time toxins are more readily carried away by reason of the increased circulation in the diseased parts.

Local treatment has for its object the ripening of abscesses and the evacuation of pus. There are several ways of attaining this, blisters and liniments of various kinds, poultices, fomenting, etc., but the best method in my hands has been the application of dry heat. This is obtained by packing the throat with dry tow or wool and retaining it there with a special hood. It has the advantage of being labour saving—a great consideration when treating large numbers of animals—continuous in action and therefore quicker than any other method. All that is required is daily adjustment of the packing material, which can be done at the time of the daily inspection. When the abscess bursts, which it does after two or three days, the tow is replaced daily after cleansing the parts till the wound heals. Local surgical interference is seldom necessary.

In all cases of strangles there is a varying amount of prostration, and in some a loss of condition, from which recovery, particularly in young animals, is slow. In acute cases the period of convalescence is exceptionally long. To appreciate this fully, one should ride the same horse before and after the disease and note the want of vitality in the movements and the rapidity with which they tire, long after all outward symptoms have disappeared. There never should be any hurry about returning horses to duty after recovery, for in addition to the danger of their being carriers of the disease, there is serious risk of joint disease supervening on even the most ordinary work, and if there are any suspicions of the wind being affected, it is a sure way of firmly establishing it. An ideal way of dealing with horses after recovery is to give them a run at grass, but this is not always possible, so one has to make the best of things by advising suitable food, the lightest of exercise and strict attention to hygiene.

Vaccine Therapy.

Encouraged by the hopeful results of vaccine therapy in abortion of cattle, I have made a vaccine on similar lines, and used it with most encouraging results in the treatment of the disease. After isolating and fortifying streptococci from strangles pus, by passing them through white mice and growing them on inspissated blood serum at 37 C. for 24 hours, large flasks (500 c. c.) of 10 per cent. serum bouillon are inoculated and incubated for one month. Six per cent. sterilized glycerine is now added, after which it is exposed in an incubator at 60 C. for two days over unslaked lime. This kills the organism and causes evaporation of the culture down to a thick paste, which is dissolved in half per cent. carbolic acid and sterilised distilled water, and brought up to half its original bulk, which makes it thin enough to pass through an ordinary hypodermic needle. It is now run into 5 c.c. sterile tubes, hermetically sealed and stored ready for use. This material which I have called *Strangline*, was injected subcutaneously into healthy animals in doses varying from 1 to 10 c.c.

The horses receiving the maximum dose had a large local reaction lasting four to five days, and a thermal reaction lasting two days, so it was decided to use it in 5 cc. to 10 cc. doses in the treatment of the disease.

The following are a few examples of the cases treated:

CAPTAIN H. E. GIBBS' CASE.

Case I. Catarrhal fever (atypical strangles). Admitted 4th March, 1910, with bilateral nasal discharge, impaired appetite, and a temperature which varied from 103 to 104 up to March 7th.

March 7th. Strangline 10 c.c. injected.
9th. Animal feeding better, and brighter. Nasal discharge visibly decreased.
10th. Strangline 10 c.c. injected.
11th. Nasal discharge of a watery nature.
17th. Animal completely recovered.

Case II. Chronic catarrh, Has been coughing and discharging from nostril since Feb. 5th. Various remedies have been tried with negative results.

March 20th. Strangline 5 c.c. injected.
23rd. Discharge from nostrils and cough almost disappeared.
30th. No cough or discharge from nostrils. Discharged cured.

CAPTAIN H. E. GIBBS' CASE.

Case III. Strangles. Admitted Feb. 20th, 1910, with bilateral abscesses and nasal discharge, and a temperature varying from 103 to 104. Abscess ready to burst on March 7th.

March 7th. Strangline 10 c.c. injected.
9th. Right submaxillary abscess burst. Nasal discharge visibly less. Temperature 101.
10th. Left abscess burst. Nasal discharge ceased. 10 c.c. strangline injected.
15th. Abscess wounds nearly healed.

18th. Another abscess forming left submaxillary region. Animal off feed. Nasal discharge recommenced. Strangline 10 c.c. injected.
22nd. Nasal discharge much diminished and watery in character.
24th. Abscess burst.
26th. No nasal discharge.
April 2nd. Abscess healed.
4th. Discharged cured.

CAPTAIN H. E. GIBBS' CASE.

Case IV. Irregular strangles. Admitted Jan. 14th, 1910. A severe case. Made no progress at all under ordinary treatment, and on Feb. 10th the horse had 16 abscesses discharging pus in the region of the throat, face and chest. He was feeding very indifferently and was much emaciated and very weak, so much so that it was decided to apply for a board to consider the advisability of destruction.

Feb. 11th. Strangline 10 c.c. injected. Result—discharge visibly diminished, animal brighter, and feeding better.
16th. Strangline 10 c.c. injected.
24th. Strangline 10 c.c. injected. Nasal discharge stopped, and all abscesses healed, except three small ones. Appetite excellent. Horse much brighter.
March 1st. Strangline 10 c.c. injected. All abscesses healed.
7th. Strangline 10 c.c. injected.
14th. Small abscess in temporo-maxillary region forms and bursts on 15th.
18th. Abscess completely healed. Horse cured.

Case V. Irregular strangles. This horse was used for serum production, and developed an abscess in the jugular groove on Dec. 9th, 1909. This was opened on Dec. 11th. It never completely healed, and a second one developed on Jan. 1, 1910. After it was opened all kinds of remedies were tried, but the abscess continued to discharge.

Jan. 30th. Strangline 1 c.c. injected. No appreciable effect.
Feb. 5th. Strangline 5 c.c. injected.
9th. Abscess healed and gave no further trouble. Discharged cured.

CAPTAIN A. J. WILLIAMS' CASE.

Case VI. Irregular strangles with temperature 105.2, submaxillary swelling, nasal discharge and sore throat. From February 11th to 16th, the swelling extended to the temporo-maxillary articulation and to the face. Abscess opened on right side on the 16th, submaxillary abscess on 17th, temporo-maxillary and five small abscesses on 19th. At this time there were 19 abscesses on various parts of the face and throat.

Feb. 23rd. Strangline 5 c.c.
25th. Abscesses much healthier and discharge less.

26th. Two fresh abscesses in right sub-parotid region, opened on 27th.
28th. Abscesses healing rapidly very little discharge from them. Large sub-parotid abscess opened.
March 1st. Abscess on right eye opened. Strangline 5 c.c. injected.
14th. All abscesses except two healed. A deep seated abscess above right eye noticed. Strangline 5 c.c. injected.
15th. Eye abscess burst.
22nd. Symptoms of pneumonia.
26th. With exception of slight serous discharge from above right eye, all abscesses healed.
31st. Symptoms of hydrothorax noticed. Animal got gradually weaker, and died on April 5th.

Post-mortem examination revealed extensive pleurisy and pneumonia, and a large mediastinal abscess containing one quart of pus.

Case VII. Irregular strangles. This horse was being hyperimmunized for serum production, and developed irregular strangles on December 6th, 1909. Abscesses formed in the jugular furrow, the left parotid region, the near shoulder, the withers, the near buttock and the off stifle, and these either burst or were opened and the pus evacuated. The horse lost a lot of condition and became so weak that he was unable to stand and had to be put in slings.

Jan. 31st. Strangline 10 c.c. injected.
Feb. 4th. Jugular, wither, and thigh abscesses healed.
5th. Strangline 10 c.c. injected.
8th. Stifle and parotid abscesses healed.
14th. 10 c.c. Strangline injected
18th. 20 ,, ,, ,,
24th. 10 ,, ,, ,,
28th. 10 ,, ,, ,,
Mar. 4th. 20 ,, ,, ,,
8th. 8 ,, ,, ,,
14th. 10 ,, ,, ,,

The shoulder abscess continued to discharge to the end although the quantity then was infinitesimal.

23rd. As the horse was still unable to stand, he was destroyed on the recommendation of a board of Veterinary Officers.

Autopsy—Extreme emaciation, a small deep seated abscess with tissue necrosis on the near shoulder, a large deep seated abscess in the near hind quarter, containing two quarts of fœtid inspissated pus. The left parotid gland was enlarged and indurated as the result of an abscess. Other organs healthy.

Strangline may be injected into the side of the neck or into the loose connective tissue in the pectoral region. I prefer the neck, as the local reaction can be seen better here. This varies in size from a pigeon's egg to one resembling a positive mallein reaction in glanders.

It disappears in the course of three or four days, and seldom gives any trouble. I am unable to say why it varies as it does.

Strangline has no appreciable effect on the temperature, which depends more on abscess formation than anything else in this disease.

Cases 4, 5, 6, and 7, are evidence of its value in the treatment of malignant forms of the disease. The life of case 4 was undoubtedly saved by its use, whilst the others show that it exerts a healing influence on strangles abscesses far superior to any recognised form of treatment. On the other hand cases 6 and 7 indicate that it has no effect on the prevention of abscess formation or on their resorption.

The quantity and quality of the nasal discharge was altered in cases 1, 2, and 3. This points to its value in the treatment of the atypical forms of the disease (so-called cases of catarrhal fever and sore throat) and protracted recoveries characterized by persistent nasal discharge, cough and general unthriftiness. As nasal discharge is one of the principal vehicles of the contagion, any remedy which reduces the quantity or gets rid of it quickly will materially help in preventing the spread of the disease. There is a further effect it is not unreasonable to hope for, viz., that horses so treated will not harbour the streptococcus in the nasal mucous membrane after recovery, and be a source of danger to others. Its action on the nasal discharge indicates that it might have a bactericidal action on the organism in this situation, but only an extensive use of the vaccine and careful observation will settle this.

The above results were obtained from the first lot of vaccine made, and as they were so promising a much larger quantity was made the second time. Unfortunately I had no shallow trays large enough for evaporating such a quantity down quickly, and instead of this process taking two or three days, it took over a week. This vaccine gave anything but good results and had to be destroyed.

There is still much to be learned regarding the size and frequency of the dose, and it may be there are stages in the disease when it is not advisable to use a vaccine. Careful clinical observation will decide these points.

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DISCUSSION.

Sir JOHN M'FADYEAN : In speaking yesterday on another subject I offered to condole with the essayist, Mr. Wilson, on the fact that he had been obliged to write a paper regarding a disease that appears to be rare in this country, or perhaps even non-existent, but no such sympathy is due to the writer of the paper that we have now to discuss, because there is no lack of strangles in this country, and there is no veterinary surgeon who has been for any length of time in practice who has not seen plenty of cases. Although the disease has been known from the earliest times, and although it has been the subject of repeated investigation and has got a very extensive literature, I am afraid it cannot be said that our knowledge with regard to it is in all respects complete. I ought to congratulate Captain Todd on having, within the limits allowed to him for a paper, presented a very good account of existing knowledge and information with regard to strangles, and his paper has the further merit that he has laid before us information with regard to treatment and prevention which is new. I propose to direct my own remarks principally to the question of the etiology of strangles. Using that word in its widest sense one might here raise the question whether strangles is a purely contagious or infectious disease, or whether it is ever sporadic. I do not suppose that at the present time there is much, if any, difference of opinion with regard to that point, but, if I am not mistaken, on a former occasion when strangles was discussed by this Association, the view was put forward that strangles is sometimes sporadic, arising independently of contagion. It may turn out that there are members here present who still believe that that is the case. I will content myself by saying that I firmly believe there is no such thing as a case of strangles of sporadic origin. We must all admit there are many cases of strangles met with in actual practice which cannot be shown to have arisen from contagion, but on the other hand I know of no published instance which would compel one to admit that the disease had arisen independently of contagion.

I want to say a word or two about the etiology of the disease in the narrower sense. For some years after the publication of Prof. Schütz's classical paper on the etiology of strangles, it was pretty generally thought that almost the last word had been said on the subject of causation : in other words, the view was adopted that beyond any doubt or question the streptococcus described by Schutz was the cause of the disease. Within recent years doubt has been cast upon that notion, and I confess I share those doubts. At the time when Prof. Schütz's investigations were first published one did not see there was any loophole to deny the conclusion that the streptococcus was the actual cause of the disease, but at the present time one can see that neither Prof. Schutz nor anybody else has actually demonstrated that the streptococcus is the cause of strangles. Prof. Schütz showed that in the suppurative lesions in strangles there is constantly to be found a streptococcus, and in a great many cases, but by no means all, that streptococcus exists alone in the lesions in a state of purity ; he cultivated this streptococcus artificially, and described its cultural and staining characters, and showed that sub-cultures of this organism were pyogenic, capable of producing suppuration in a variety of animals. He also showed that the streptococcus was capable of producing diseases in horses, but it is open to anyone to maintain that that falls considerably short of proving that the so-called strangles streptococcus is the actual cause of strangles. What one is entitled to demand in the case of any investigation purporting to establish the connection between an organism and a disease, is that with pure cultures of the organism, if it be a cultivable one, one can reproduce the natural disease with all its essential characters. Now one essential character of strangles is that the disease is contagious. The mere fact that the strangles streptococcus produces suppuration is not sufficient to prove that it is the cause of strangles. What would satisfy one that the streptococcus is the actual cause of strangles would be a series of experiments in which it was shown that by using a culture of the streptococcus one could infect a horse, and that the artifically infected horse would then hand on the disease to other susceptible horses placed in contact with him ; but so far as I am aware that has never been done, and I very much doubt whether if the experiment were tried it would have a positive result. It is at any rate very much to be desired that investigations of that nature should be undertaken. Unfortunately they would be rather expensive, because in order to be sure that one was not dealing with horses already immune from strangles one would probably require to employ young or expensive foals or colts.

I admit that in the absence of such crucial experiments as these one might find in another direction evidence to convince us that the streptococcus is the actual cause of strangles. If, for example, it should be shown beyond question that one can immunise horses against the natural disease with the streptococcus or its products, then I think that that would be proof that the streptococcus is the actual cause of the disease—but I shall touch upon that question later on.

In the next place, I should like to be allowed to say a few words with regard to the method of infection. If I am not mistaken, Prof. Schütz in his paper expressed the view that the point of entrance of the strangles streptococcus is usually the air passages—in other words he believed that infection usually is by inhalation. In reality I do not think there is any positive knowledge with regard to that point, but I notice that Captain Todd in his paper does not favour the view that inhalation is the common method of infection, and is rather inclined to the view that the common method of infection is the ingestion of discharges containing the streptococcus. I do not think he himself possesses any knowledge derived from rigid experiments which would compel one to adopt that view, but he attempts to throw some discredit on the inhalation theory of strangles infection by saying that recent research has shown that this method of infection in other diseases of the air passages is very uncommon. When I read that I wondered what other diseases of the air passages this referred to. I believe at the present time absolutely erroneous notions are held widely by veterinary surgeons and medical men regarding the relative vulnerability of the digestive tract and the respiratory passages to infection with certain bacteria. In consequence of a paper that was read in this country about two years ago by Sir William Whitla, in which he gave an entirely one-sided view of the question, the opinion has largely been accepted that the inhalation theory as applied to tuberculosis has been absolutely discredited. I do not, of course, wish to go into the question of the method of infection in tuberculosis, but I will venture to say—and I am quite willing to supply the evidence to anybody afterwards—that it is the ingestion theory of tuberculous infection that at the present moment is absolutely discredited. So far from it being a fact that animals are more readily infected with tuberculosis by ingestion than by inhalation it has been shown that the vulnerability of the respiratory passages to the tubercle bacillus is at least nineteen thousand times greater than the vulnerability of the alimentary canal, and the truth is that we shall have to reconsider in the light of these recent investigations whether we have not been too hasty in concluding that inhalation is not, after all, the common method of infection in strangles. At any rate, until experiments have been conducted to show what is the minimum amount of strangles infection that will certainly infect by the air passages, and the minimum amount that will certainly infect by the digestive tract, our opinion as to what is the common method of infection in strangles should be suspended. In truth, however, I do not think anyone could pretend that it is a matter of very great importance whether a horse is usually infected with strangles by ingestion or inhalation, but everyone will admit it is desirable that we should know what is the common method of infection. It would certainly be unfortunate if one should wrongly suppose that a horse stands no danger of contracting strangles unless he has an opportunity of ingesting the strangles streptococcus or the virus of the disease ; and I would like to ask Captain Todd whether he thinks there is no possibility, for instance, that strangles may be spread in the shoeing forge or in other circumstances of contact that make inhalation of the virus probable but which perhaps would not admit of the ingestion of the organism.

Another point upon which I confess I do not see eye to eye with Captain Todd is regarding the influence of weather and other conditions that tend to depress the general vitality. I would not deny that if a horse is in a condition of general debility his susceptibility to strangles infection may be increased, or his power of resistance be to some extent reduced, but I am not satisfied that any young horse that has not previously had an attack of strangles is capable of resisting infection, no matter how vigorous his condition may be at the time when he is exposed to infection, and I should like to suggest to Captain Todd that the table which he has printed (page 100) is not so convincing in this connection as he supposes. We are asked to infer from that table that the exposure of young horses to severe winter conditions is highly favourable to the development of strangles; and of course if one does not think whether there may not have been other explanations the chart is very striking, inasmuch as it shows that in the month of February there were no fewer than 120 cases of strangles in the stud, whereas the cases rapidly declined during March and April, and the disease had almost disappeared in the months of May and June. But I would suggest to Captain Todd that the cases fell off in the spring months simply because remounts were not then being brought into the stud, and nearly all the animals had then had strangles. The number of horses in the depot in October was 60; 20 were introduced during the following month, making 80; but the maximum number in the stud was in January and there appears to have been no horses introduced after January at all. What I suggest is that if 100 young horses had been introduced monthly the chart would have been of quite a different type, that the cases of strangles would have maintained a level line instead of falling.

It will be very good news to the profession to hear that the experience gained in the army amongst army horses goes to show that it is possible to obtain an effective anti-strangles serum. The question whether such an effective serum can be manufactured or not is one that I have followed with interest for a good many years. I have endeavoured to keep myself posted as to the experience in this connection abroad, and I confess that I have not been hitherto able to form a very decided opinion as to whether any of the anti-strangles sera on the market were of use or not. I was very much impressed during the past year with the experience of an author whose paper is referred to by Captain Todd, namely, Desoubry, but what one would like to have is information showing the protective effect of strangles serum on a large series of horses afterwards exposed to contagion along with an equal number of unprotected horses. In any such experiment it is eminently desirable that the number should be a large one. I believe it is unlikely that we shall ever get an experiment of that kind conducted in this country outside the army, and we shall all be glad to hear whether Captain Todd is in possession of any such statistics. I hope that it may be possible to continue any such experiments as may already have been carried out in the army in order that we may know for certain whether an effective anti-strangles serum can be manufactured or not.

With regard to other methods of protecting against strangles or curing strangles, I should like to refer to the use of killed cultures, or the so-called Strangline, which Captain Todd has been manufacturing and using. I earnestly hope that an enlarged experience will confirm him in his present opinion that this is really a valuable method of treatment, but I hope he will not be offended with me if I say that I do not think the small number of cases he has given us justifies a very strong opinion one way or the other. No strong opinion would be justified in the first place because the cases are too few to prove anything, but I would like to suggest to Captain Todd that the cases really prove a little too much. I always understood—and I still entertain the opinion—that when one uses killed cultures or living cultures with the view of influencing the course of a disease of which an animal is already suffering, you cannot get any immediate benefit. The rule, in fact, is that when you introduce either dead or living cultures into an animal you get what is called a negative phase. You actually depress for the time being the animal's defensive powers. But that apparently has not been the case in Captain Todd's experience, because you will observe that in some of these cases the thing appeared to act like a charm. The day after the vaccine was administered, or the second day, there appeared to be a manifest improvement in the course of the disease and in the suppuration. It is in that respect that I think the cases prove a little too much. I do not say that this strangline is not a valuable preparation, but I think a larger series of observations must be made before favourable results ensuing in an animal treated can be ascribed to the strangline. One knows that the crisis of the disease in different cases of strangles is very different, although the animals may have been treated in the same way, or may not have been subject to any treatment at all.

There are several other points in connection with this paper that are of very great interest, but I have already occupied too much time, and I am sure there are many here who are capable of taking up the discussion and making criticisms from the point of view of practitioners which will be much more to the point than any I could make.

Mr. Dunlop: Mr. President and gentlemen, I do not know that I can speak with authority on the subject of strangles; it is so long since I, in a large measure, severed my connection with the veterinary profession. I have very few remarks to make except to support the statement which Sir John M'Fadyean has made. I do not believe from my experience over twenty years ago, for thirty years or so, that strangles is sporadic. The question is whether it is contagious or infectious. It is very difficult to define the difference between contagion and infection. In both cases it is merely a transplanting of the microbe, the seed, or the spore, to another soil adapted to its reception. I might ask the question, does dessication destroy the microbe? If it does, then I would say it is contagious only. With regard to tuberculosis, I was very much surprised when I first heard that it was entirely due to or communicated by inhalation. I did not see the object in preventing people from spitting on the pavement, because the contagion must be desiccated and get into the form of dust before it can be inhaled. We all know that stone masons are particularly subject to tuberculosis, and we know very well that the dust from the stone does not cause tuberculosis of itself. No doubt—this is the only reasonable explanation which occurs to me; there may be some other—the dust causes a great amount of irritation and cultivates the soil, the same as an ordinary cold setting up inflammation of the bronchial tubes makes the soil, as it were, better adapted to the reception of the germs. I am very pleased to hear Sir John say that the trend of opinion now is rather in favour of tuberculosis being communicated by dust. I am not able to say positively whether strangles is communicated by dust or not, but I have seen cases where it was communicated without any direct contact. It was quite true that the place may not have been thoroughly cleansed out and contagious matter may have remained in the manger, but I am quite positive that I have seen it in many cases communicated without any direct contact. I have had considerable experience in the prevention of disease, having been the first inspector appointed for the City of Belfast and a very large surrounding district when disease was very rife, and I have had considerable experience in stamping out contagious diseases. I think the records of Dublin Castle will bear me out in saying that I was fairly successful. It was not so much through my own endeavours that I was succcessful, but it was because I had the sympathy and support of all the dairy keepers in Belfast and the surrounding district, and nothing occurred which was not immediately communicated to me, for the simple reason that in my practice I was pretty well known amongst them personally and I always studied their interests, and they thought it was to their interests to communicate to me at once. I have always thought that we did not pay enough attention to the prevention of dust, and I have thought that, with regard to the human subject, even handkerchiefs and the outer coverings of nurses should all be treated by some disinfectant. I am afraid I am getting away from the subject, because I am not able to say positively whether desiccation actually destroys the germ of strangles or not, but I am quite positive it can be communicated indirectly. I have no faith in the theory of the disease being sporadic. I do not know I have anything to say with regard to treatment, and I certainly do not believe that it is necessary for a horse to have a cold or to be run down in any way to develop strangles. I have seen strangles breaking out amongst young horses which were in the pink of condition and in perfect health, in all seasons of the year.

Col. STEELE : There are two points I should like to offer for consideration. One is whether it is advisable to introduce this poison into a regiment or a stud at all with the idea of preventing the greater evil afterwards. I think we are carried away in many cases by the fascination of the preventive means—which are admirable in their way—of inoculation, and the use of sera, and so on. I also wish to refer to the depressing effect, as stated by Captain Todd, of exposure. I am inclined to contend that exposure is rather antagonistic than favourable to the development of strangles. I will make my story in support of this latter assertion very short. It is going back into ancient history I am sorry to say, but many years ago I assisted in the purchase of 35 remounts for the 12th Lancers. The 9th Lancers purchased exactly the same number from the same source, all long tails, three or four years old, which had been living a life of exposure. They came by the same train to Dublin, crossed in the same boat, and were conveyed by the same train to Aldershot. On arrival at Aldershot our horses—and when I say "our" I always speak with affection of the old regiment, and delight in it—were stationed with the regiment in the West Cavalry Barracks, which those who know Aldershot are aware are on low ground. The horses of the 9th Lancers were stationed on the hill in the South Cavalry Barracks, and therefore would be presumably more favourably situated as regards ventilation and drainage. I had, I am proud to say, the confidence of my commanding officer at that time, and he allowed me to do exactly what I liked with these remounts. I was laughed at for my extreme views as to the value of God's air, but I have maintained those views ever since. Our horses were put in stables with an abundance of room; we kept the doors and windows open day and night—this was in October. I went to the extreme of exposure, I might call it. The 9th Lancers acted, as it was presumed, more judiciously. They went round at night to see which way the wind was blowing so that the doors and windows should be closed to windward, so that the horses should not be exposed to that immensely exaggerated source of disease, a chill. The result was that we had not a single case of strangles amongst the 35 horses, whereas the 9th Lancers had hardly, if any, exceptions. In the Spring our young horses were trained and put into the ranks, and the commanding officer had the pride of coming out stronger than our neighbours the 9th Lancers, because their remounts were only then beginning to be trained in consequence of the drawback which they had sustained by attacks of strangles. They lost one or two. I have no data to go upon, but I state that some of them were not efficient in their respiratory apparatus after their experience of strangles.

Mr. Ainsworth Wilson: I should like to make one or two short remarks on the question of strangles. With regard to age we are told, and rightly so, that age is a well-established predisposing cause, and a quotation has been made that "Viborg and Hertwig observed that 70 per cent. of cases occurred in horses under five years of age." I think we agree with that. At the same time in civilian practice, in some outbreaks we find that the disease is more or less confined to old horses. Perhaps in such stables young horses are not present, but my point is that old horses are largely affected with the disease in certain outbreaks ; and in some of my cases they have been more severely affected than the young ones—and also it is unnecessary for the vitality of the old or young horses to be lowered. That point has already been referred to. But at the same time we all admit, I think, that anything which reduces the vitality—disease or other causes—predisposes to the infection of strangles and other diseases. I want to ask Captain Todd with regard to desiccation killing the organism after three weeks, whether the organism can retain its vitality outside the body in a moist condition for a longer period, because I have had outbreaks in which I have considered that the infection has remained on the premises for some months, probably in damp corners and so on, in the absence of sunlight. I should like to know how long Captain Todd considers it may retain its vitality under favourable conditions outside the body. A point which I think is of great importance is the fact that a horse may recover from the disease and be to all intents and purposes a sound horse, and be at work for a considerable time, and yet after an appreciable length of time will infect other horses. Captain Todd referred to this point, and I should like to quote an instance, though perhaps not a conclusive one. A client of mine, a farmer, had two farms separated by a distance of three or four miles. There was no communication at all between the two farms. An outbreak occurred on the first farm on which were seven horses. When called in I found two affected, a two year old and an old mare nearly twenty. The two year old was affected with ordinary strangles, with an abscess formation in the jaw and throat—pretty bad, but still, a case of ordinary strangles. The old mare was affected in a similar way, but developed irregular strangles, and we had some trouble to pull her through. Of the seven horses which were on that farm six were affected more or less. After making a complete recovery these horses were put to work, and three months after the recovery—not after the beginning of the disease, but after the recovery—one horse was removed from this farm to farm No. 2. There had been no communication at all between the two farms. The owner had ridden across once or twice, but he had never gone near the horses; in fact, he remained near the field and looked over the gate. I do not think there was any chance of the horse he was riding conveying the infection in any shape or form. As I say, three months after these horses had been put to work, one was removed to farm No. 2. Within eight days, of the six horses on farm No. 2, one became affected, and subsequently three more. I can only attribute the second outbreak to this recovered horse being removed from the first farm.

With regard to atypical strangles, these cases occur, I think, largely in horses which have a second attack of the disease. I agree entirely with Captain Todd that where we have an outbreak of strangles all cases of catarrh, whether associated with fever or not, should be treated as strangles. I do not think anyone will quarrel with the incubation period. The infection is conveyed in various ways, and it is surprising, in country practice at any rate, how many outbreaks we get in which we cannot trace the source of infection. The disease seems to arise spontaneously. Sometimes there are no cases of strangles at all in a district, but one gets an outbreak on a farm to which no horses have been brought and where the disease has not existed perhaps in the county as far as one knows at the time.

The point of greatest interest to me in the paper is that dealing with prevention and treatment; I refer to the use of serum and the use of dead cultures. Serum in this country has been used very largely, but unfortunately we have no reliable statistics in regard to it, that is to say, that where results have been published the number of cases does not warrant one in coming to a definite conclusion. In one outbreak I had the opportunity of testing, as I thought, the effect anti-streptococcic serum. This was a Shire breeding stud consisting of twenty-five animals, foals, yearlings, two-year-olds, brood mares, a stallion, and six cart horses, the latter being kept apart from the breeding stud. When called in I found several animals affected with ordinary strangles of a severe type. The disease spread very rapidly, and within a few days 16 of the 25 breeding animals were affected, beginning with the foals and working right through the stud. The stallion was not affected. The owner was very anxious, and fortunately expense was not a consideration; at any rate I was allowed a free hand, and I obtained a large quantity of serum. There was good nursing, plenty of air and ordinary hygienic treatment, but we relied principally on serum. To begin with I injected every animal with the idea of prevention, and I may say that after using the serum no more animals became affected. I do not know whether the serum had anything to do with that. I used the serum, however, principally in the treatment of the 16 affected animals. Of those 16, four, two foals and two two-year-olds, were very badly affected, and these animals received repeated injections. The other affected animals received only one or two; all received one. With regard to the four badly affected animals, one especially I want to say something about. This was a foal. She had developed abscesses in the throat and under the jaw, and there was one on the shoulder and one on the flank. Unfortunately the throat was very bad, and there were premonitory symptoms of pneumonia, with a very high temperature varying between 105° and 107°, no appetite, great depression, discharge, and all the rest of it. This animal looked like dying; in fact, the owner gave her up. I used the serum in

10 cc. doses throughout, injecting one dose for the first three days and then injecting a dose every second day until the 10th day. I may mention there was no improvement for four or five days; she got weaker and it was very difficult to give nourishment, and really I thought we were going to lose her. But after the fourth or fifth day the improvement was very rapid. The temperature went down, the appetite returned, the abscess formation continued, but to a slighter extent, and the discharge was less. The whole condition of the animal improved rapidly. In the other three affected animals the improvement was also marked after a few days, and I concluded that the serum had certainly had a beneficial effect.

Major Gen. SMITH: Mr. President and gentlemen—Those of us who are charged with the responsibility of the veterinary care of a large stud of horses, cannot possibly look other than with a feeling of apprehension upon the introduction of a disease like strangles, especially amongst the young stock. When I became responsible for the health of the horses of the Army, strangles was one of the diseases which I felt we ought certainly to take in hand and see whether anything could be done to improve the treatment, and if possible to reduce the mortality, though at no time was the latter high. What troubled us particularly was the inefficiency which resulted from a large number of horses being laid up at the same time, because we only buy at certain periods of the year, and that meant a very large amount of sickness existed for a certain number of months in the year, and this sickness was followed by other causes of inefficiency which I need not here enter into. In consequence, I consulted with my senior officers as to what would be the best method of trying to get at a more exact knowledge of the trouble which existed, and I subsequently found in Captain Todd an enthusiastic observer. who was anxious to proceed abroad – where I felt we might pick up some information—to gather what knowledge he could and to bring it back to us. This forms no part of his paper, and he will pardon me in referring to it, but he undertook at his own expense to visit certain capitals in Europe and collect all the information he could. The result was that he was able to tell us that certainly some of the sera which were then on the market were quite unreliable. He also brought back some very definite information. He had been to Rotterdam, and he had had an opportunity there of going very carefully indeed into the methods which were adopted at the State Serum Institute, where they gave him every facility for examination and investigation, and had also shown him some of the results derived from the inoculation of the remounts in the Army. These results appeared to us to be very encouraging, so encouraging indeed that after some little difficulty we succeeded in getting the authorities to sanction the production of serum on the same lines as those which were being carried out at Rotterdam, and Captain Todd was sent there in order to learn the methods so that we should in no way depart from the experience they had gained, and be able to produce practically the same material in this country which was being made there. I make these observations as a preface to such remarks as may follow, in order to explain to you the position in which Captain Todd stands with regard to the paper which he has produced here to-day.

I agree with Sir John M'Fadyean that there are no cases of sporadic strangles, but how we are to account for outbreaks where there is no obvious cause of contagion I really do not know. I believe much the same difficulty existed with regard to foot and mouth disease years ago, and it was found then that the human agency played a very important part. It may yet be shown that human agency may play a part in the dissemination of strangles. I cannot at all agree with Captain Todd that hard work, or cold air, or sudden changes of any kind are important irrespective of infection. As to repeated attacks of strangles, I know nothing whatever about them. I have seen horses attacked a second time, but so very, very few that I should think probably not more than two or three in my lifetime. I would ask—and I feel sure there are many in this room who would be able to enlighten us on the point—whether it is a fact, as stated by Captain Todd, that strangles is rarely seen in the stable where buckets are employed for the watering of horses instead of water troughs. I cannot think it is right. If it were, nothing would be easier than to block out strangles from a large body of horses, although again the qnestion of the individual watering of horses from buckets would also be a difficult matter. I do not see how one is going to prevent the buckets from being interchanged.

Sir John M'Fadyean mentioned a very important point indeed, namely, as to how the strangles organism was introduced into the body, and he threw some little doubt upon ingestion as playing any important part. I may say that before we adopted the Rotterdam serum we used, I think it was four, different sera obtained from different parts of the world, more particularly from the Continent of Europe, and in order to test whatever value they possessed, I endeavoured, in those animals which we believed might possibly have been protected as the result of the injection of serum, to try and infect them subsequently by the introduction of strangles virus. The method adopted was that of ingestion. With regard to those sera, which proved to be utterly worthless—and I am afraid some of them are still on the market—the animals so treated and subsequently fed on strangles virus mixed up with their food, almost invariably contracted the disease, from which I judge that ingestion must be a very common cause of infection. I cannot give you the number; I was not prepared to answer the question to-day or I should have looked the figures up, but the numbers were sufficiently large to impress upon me very forcibly indeed what an important part ingestion must play in the spread of the disease, and how very desirable it is in dealing with young horses, especially those which are ailing, to throw away the contents of whatever they have left in the manger, in order to avoid any possible chance of infection being conveyed to healthy animals. There is one point which I do not think was very forcibly brought out in Captain Todd's paper, or perhaps I may have overlooked it, namely, the frequency with which occult strangles exists in horses. We recognise strangles when it is "open," when we have an abscess generally in a definite position, and when none of us have the slightest difficulty in saying that the horse has strangles. But I feel perfectly sure that, in spite of all the natural caution which one attains as the result of age and experience, there are a very large number of horses affected with strangles which have no external abscess at all—that the abscess may form in the pharynx or the post-pharyngeal region, associated with a catarrh; there is, at any rate, a discharge from the nostrils, fever, and other indications of serious ill-health. Those cases may have no external manifestation at all, or they may have the slightest possible swelling or thickening in the intermaxillary space, which may disappear in a day or two. We are waiting in those cases for the abscess to form between the jaws which never forms. It would be very wrong indeed, I think, to say that those animals have no attack of strangles. I feel perfectly satisfied that they are going through the disease just as thoroughly and just as effectually as if they had the abscess forming in the intermaxillary space. It is a very important point indeed in practice, so much so that I impress upon my officers to treat all cases of high temperature with a muco-purulent discharge from the nostrils as if they are highly infected and as if they possessed external signs of strangles, so that there should be no possible doubt. I think if that practice were followed the spread of infection would be partly controlled.

The relationship between strangles and pneumonia has not been dealt with by any previous speaker, and I am not sure whether Captain Todd lays sufficient emphasis upon it. Some few years ago I placed myself in communication with Sir John M'Fadyean on the matter of the frequency with which pneumonia, pleurisy, and strangles existed in a particular remount depot, of which at that time I had charge. I did not connect the two diseases in any way. It did not occur to me to think that pneumonia and strangles could in any way be associated, probably owing to the fact that I was so very ignorant of bacteriology and of micro-organisms in general. It was Sir John, however, who told me that organisms might be found or might exist in either pneumonia or pleurisy which could not be distinguished from the organism which at that time was associated with strangles. Immediately I was placed upon the track, and I was able by clinical observations to frequently associate the two conditions. I feel perfectly certain, from clinical observation that the two diseases are intimately connected, so much so that I was going to hazard a guess as to the proportion of cases of pneumonia which follow strangles, but perhaps I had better not. At any rate it is so very common that it is one of the points to which I believe those who are in charge of young horses cannot possibly pay too strict attention.

Sir John has asked a very pertinent question indeed—whether an effective anti-strangles serum can be produced. It is the crux of the paper which is before us this morning. I was not able to give Capt. Todd, for the purpose of this paper, certain figures which I propose to give you now. I hate to clothe myself with red tape or to appear in an official capacity, but I could not give them to him for the reason that they belonged to an Official Report, and they could not appear in the Proceeding of our Association until they had appeared in the ordinary Government Report. But I propose, with your permission, sir, to read to you the Annual Report, which at present is in the press, which deals with this question of strangles and serum.

"In the last Annual Report attention was drawn to the all important question of strangles and its prevention by inoculation. A brief account was given of some observations made with different varieties of sera, and it was stated that the results were encouraging. After many observations it was generally felt that the only serum to which any value could be attached was that manufactured by the State Veterinary Laboratory in the Netherlands, and through the courtesy of that Government and the kindness of the Veterinary Director, Dr. Poels, an officer (who was Capt. Todd) was sent to Rotterdam to study the question of serum production. It is unnecessary to relate here the somewhat elaborate technique necessary for its manufacture, but it suffices to say that following on the Rotterdam lines we have produced a serum which it is hoped will prove of value. The matter being experimental the output is limited, and in the meantime we are supplementing deficiencies by purchases in Holland. It seems a simple question to be decided whether a given substance does or does not act as a preventative of disease. As a matter of fact it is a complex problem, and exact information cannot be at once obtained. It took ten years to establish the value of the anti-diptheritic serum, while the protection against typhoid fever in man is still far from settled. As an example of one of the difficulties attending the collection of information, it is only necessary to point out that no matter how favourable a serum may be in its action, time is required after its introduction for the tissues of the body to be rendered resistant. An animal inoculated to-day cannot be protected against an infection contracted a week ago or even of that to which it was exposed yesterday. Similarly some days—we do not know yet how many—must be expected to elapse after inoculation before infection subsequent to inoculation is rendered harmless. Link up with these the inevitable differences of human judgment, and we are then in possession of the chief elements which create doubt and render investigation not only difficult, but even perhaps less exact than the figures obtained would warrant us in supposing. In other words, time, and only time, can supply an answer to this important question.

"The result of inoculation during the past year was wholly favourable. Only one variety of serum has been used, namely, that purchased in Rotterdam or cultivated on the lines adopted in Holland. The following are the result of 241 inoculations carried out by the same observers and as far as possible under similar conditions in the remount depot at Woolwich. The total number inoculated was 241. The number of horses subsequently affected with strangles was 31. The number of deaths, 2. In this case 12.8 per cent. of the animals contracted the disease. The mean proportion of horses attacked in the depot during the four years 1906 to 1910 was 12.5 per cent. From this it might appear that inoculation was of no value as a protection, but an examination of the figures shows that out of the 31 horses affected, 21 of them were either affected at the time of inoculation or within seven days of the operation being performed; while ten were affected between the 11th and the 34th day after inoculation. It is considered that only these ten should be regarded as failures to protect. The 21 cases occurring up to the 7th day may safely be regarded as infected prior to inoculation. Still, the fact is indisputable that certainly four per cent. and probably more were not protected, and in one case infection resulted on the 30th day in spite of five previous injections with serum at various intervals. In regard to this latter observation, it is necessary to add that it should not be invested with too much importance; it is an isolated observation, but of value if viewed in its true perspective. The incubative period of strangles is still not experimentally settled; it probably lies between the seventh and tenth day, but that it may be considerably prolonged is undoubted. In the above case this horse, though not suffering from open strangles until the 30th day after inoculation, was no doubt systemically infected some time before, for on turning to the clinical history of the case it was found that a bad catarrh was recorded on the day of inoculation, and so doubtful did the case appear that the serum was repeated the following day, again three days later, and for the fourth time three days after the third inoculation, and for the fifth time a week after the fourth inoculation. Evidently, therefore, this horse was affected from the first, though open strangles did not declare itself for thirty days. In spite of these facts the case is credited as a failure for the reason that it cannot be proved that ample opportunity was not given the serum to prevent or to abort the attack.

Since the meeting the results of 239 further inoculations have been ascertained. In the official report these are spoken of as the second series:—

"In brief, we have in this second series 239 horses yielding 50 cases of strangles, and of these 50, only six can be shown to be in all probability free from disease at the time of and for some days subsequent to inoculation. Consequently in these six cases the serum failed to be of any benefit. The remaining 44 cases either developed the disease in so short a time after inoculation, or suffered from a preliminary catarrh which subsequently ran concurrently with the disease, as to lead one to believe that the serum did not work under favourable conditions. That, at any rate, is a fair conclusion to be drawn from the results, though my personal bias inclines me to regard these cases in the main as failures to protect."

"The next question to deal with is whether the serum in the treatment of the infected is of any value, and on that point there is almost a general consensus of opinion that under its influence the cases are milder, that the horses remain brighter and feed better, that the character of the discharge from the abscesses becomes favourably altered and reduced in quantity. These are valuable assets; in fact, there are some who, for good reason, consider that the protection of horses against strangles is not an unmixed blessing unless the protection is permanent. The introduction of strangles on mobilisation, for instance, must always be borne in mind, and its spread at this time could not lightly be considered. If, therefore, the Rotterdam serum helps the sick to make a better recovery, a distinct advance has been made, and on this point we are inclined to give a positive assurance, both from clinical observations and from statistical enquiry. The death rate in the Remount Depot at Woolwich has improved. Calculated on the number of admissions, it was as follows: in 1907 to 1908 it was 4.3 per cent. In 1908-9 it was 2.8 per cent. During that year several different varieties of sera were used in the treatment of the disease. In 1909-10 the death rate was 1.8 per cent, and during that time the Rotterdam serum was exclusively used. It is possible, of course, that the strain of virus during 1908-9 and 1909-10 may have been milder, but at present there is no evidence to show that the improved death rate is not due to the method of treatment conducted, and, coupled with the clinical observations of improved appetite and brightness, the credit is accordingly given to the serum.

"Another important question is the influence of serum treatment in the prevention of roaring following strangles, and on this point there appears to be good reason for thinking that benefit has been derived. Some figures might be collected which would give practical support to this view, but it is considered at present better to allow more time to elapse before forming a definite opinion regarding the wind, as roaring may follow months after an attack of strangles."

At this moment I am thinking of the fact that Capt. Todd has given you some of these figures. I did not introduce them into my official Report because I thought they were a great deal too good. I was frightened in case it was a mere accidental circumstance this year, and that probably in years to come we would have to climb down. It is far better to climb up than to climb down. The report goes on:

"In the meantime those best qualified to judge of this important question are clearly of opinion that it acts as a preventive of roaring. Efforts have been made at the Veterinary School at Aldershot to produce a vaccine for strangles with the object of hastening the cure in protracted cases of the disease. The substance produced is obtained from a pure culture of the organism of the disease and has been designated Strangline. At present the feeling with regard to the value of this substance is one of doubt, but that may be due to the fact that the question of dose has not been settled with exactitude. It may be a mere coincidence, but the first case on which this vaccine was tried was apparently, in spite of serum, in a hopeless condition from strangles and was about to be destroyed. Under strangline it made a considerable improvement and eventually recovered. Strangline takes one-third of the time to produce, and is far cheaper than serum."

I have ventured to trouble you with a statement which will shortly be published, because I thought it would enable me, at any rate, to express an opinion on the conclusion at which one had arrived after very careful examination of a large number of figures, and I felt if I read this to you it would be impossible for me to make an error in any statement which I had the pleasure of making at this meeting, and would avoid any misapprehension in the future. The position, therefore, with regard to strangline is that we are in grave doubt. Capt. Todd is, I think, far more optimistic. With regard to serum, it does not prevent the disease from occurring. It does, as far as we can see, help an animal to recover, and renders a case milder. I have to thank you for the very patient hearing you have given me.

Col. BLENKINSOP: There is one point which I think will strike all people connected with this disease, namely, that it is of great importance with regard to mobilisation of horses for active service. We have to rely to a very great extent on the civil horse population for our remounts in war. Our experience of the South African War—and I had a good opportunity of seeing the result of collecting horses there, as I was senior Veterinary Officer of remounts for some considerable time—was exactly the same which occurred a hundred years before, in the Napoleonic wars, when they collected horses up and down the country. Our horses arrived at South Africa in shipments from oversea. They were infected with this particular disease, which gave us a great deal of trouble; and there is no doubt that the horses which came from the English ports at that time suffered also from what I imagine must have been a mixed infection, because we did not get the death rate from other horses as we did from those which came from this country. If we could get immunity from any sera, it is to be hoped that the immunity will be permanent, because should it break out when we are under service conditions it would be a very serious thing. The treatment of the disease with serum, from what I have read—I have not had any opportunity of experimenting myself—has every prospect of success, but I think we should not place too much confidence in sera giving immunity from the disease. You cannot get rid of strangles altogether. I doubt very much if we will ever get rid of it. I think it should be treated as a contagious disease, and that we should take steps to limit the number of cases in our country. The importance of strangles among young horses is one which I have had opportunities of seeing in foreign countries. I have seen strangles appear in the most virulent form among young stock, and enormous numbers of animals have been lost. This happened not very long ago in South Africa, in the Orange River Colony, where studs of young horses were affected and died in large numbers. In one instance we had a number of old and young horses that came over on ship from England. Among the old horses we had very few cases; I think out of 60 horses we had four cases, but the young horses suffered to a very large extent. We kept the cases in the depot and they were treated there, and after about three weeks or a month after recovery they were distributed to other centres. There were no cases of strangles resulting from the distribution of those horses, although many of them were young horses which did develop strangles when they were subjected to infection. I do not think that when a horse has recovered from strangles and has been allowed to live in the open, it is capable of spreading the disease after about three weeks or so. That is my clinical experience from watching this disease. The incubation period of the disease, I think, as far as one can make out from clinical observation, should be placed at about ten days in the majority of cases, but fixing the incubation period is a very difficult thing.

Prof. WOOLDRIDGE: Up to the present the enormous economic importance of this disease to the country has apparently escaped the notice of the veterinary profession, or at any rate, of the public in general, and I think when one realises the loss it entails, not necessarily in the death of animals, but in the length of time they are thrown off work, we cannot be too grateful that the subject has been taken in hand in such a systematic manner as appears to have been done in the army at the present time, and our thanks are due to Captain Todd for having brought the matter before us in such an able manner. There are one or two points in connection with the paper and the discussion which I should like to refer to.

In the first place, in connection with the method of infection, there seems to be some difference of opinion as to whether infection is by ingestion or inhalation. I think the point may again escape some of us which ought to attract our attention principally, namely, not so much can it be transmitted by ingestion, or can it be transmitted by inhalation, as Which is the commoner method of transmission? In the cases that have been referred to where infection has been conveyed experimentally by ingestion, it seems to me that as in other diseases, the amount of infective material which has been given has been enormously in excess of the amount which would probably be taken in in natural circumstances; consequently I think we are apt to draw a wrong conclusion. The only thing it seems to me to prove is this—that infection may be conveyed by ingestion; it does not prove that it is the common method of infection. The position of the lesions, the symptoms that are exhibited, must be taken into account also in coming to some decision on this subject.

The condition met with in adult cases, which have been referred to by General Smith, had also attracted my attention; I refer to that condition of nasal catarrh in adult horses in the discharges of which can be found frequently a streptococcus indistinguishable from the streptococcus of strangles. The question arises as to whether we shall consider this a case of strangles or not. I believe that it is strangles. Then how is that condition of the disease set up in which you have got lesions limited to the nasal mucous membrane? To me the answer appears fairly obvious: I think it is undoubtedly by inhalation in those cases. Then, again, we have another condition to bear in mind, which is common in young horses, to which reference has not been made in the course of the discussion; I refer to the easy portal of entrance of organisms in young horses when they are erupting teeth. I cannot at present dismiss from my mind the possibility that in many cases of strangles infection is carried in that method. The opinion, I hold, then, at present, in view of the various experiments and reports that have been brought forward, and the observations which have been made, is this, that there are three methods of infection—by inhalation, which is a very important method; by inoculation, through wounds of the gum; and by ingestion in those cases where horses have been able to take up enormous quantities of the discharges from other infected animals. I do not think that it is by ingestion, at any rate in the first cases that occur in a stud, where the amount of infective material, with which the horse has been brought in contact must usually be comparatively small.

I was very glad indeed to hear the excellent results which appear to be obtainable by the treatment with serum as a preventative, but I think it is not exactly reasonable to expect immunity of a very long duration produced by that method. It is contrary to experience in the other infective diseases. An immunity produced by a serum is what is known as passive immunity, which is almost invariably of a very much shorter duration than that which results from recovery from an actual attack of the disease, or where immunisation has been brought about by the use of attenuated culture. I think the serum will be of the greatest possible benefit where a disease has made its appearance in the stud, in order to diminish the spread to the stud. The introduction of this material, strangline, probably will mark an important stage in our knowledge in connection with the disease, but I do not feel convinced at the present time from the cases that have been cited by Capt. Todd that he has obtained yet a direct curative agent. In looking through the various cases, with the exception of the first case, and noticing the course the disease has run, I have been struck with the fact that the course has not been apparently shortened from the present natural course of the affection. For example, in the second case the animal was admitted into the hospital, or at any rate had been suffering from a discharging nose, and from a cough, from February 5th to March 30th, that is, a period of about seven weeks. My observation of the disease seems to me to say that the natural course of the disease runs about five to six weeks, and here is a case which has been treated with strangline where the disease terminated at about the end of the ordinary course. Certainly, immediately after the injection of the strangline some beneficial effects were noticed in connection with the abscess itself, but apart from that it ran its ordinary period. The same applies to case No. 3 which lasted for six weeks, and the case was not discharged cured until four weeks after strangline had been injected. In connection with case No. 4 the same thing again occurs. Certainly it was a very severe case, where 16 abscesses were present. The course in that case ran from eight to nine weeks, and there were five weeks from the time of the injection of strangline before the case was discharged cured. In case No. 6, where death occurred, from six to seven weeks was the time of admission, and five weeks after the injection of strangline. It does not appear there was much beneficial result in that case beyond some improvement in the abscess itself, which, of course, is not the important thing. It is certainly of some importance, but the main thing is that we should be able to get the horse completely recovered in a shorter time if we are to attach much importance to this particular method of treatment. The same thing occurs again in connection with case No. 7, although perhaps that is hardly a fair one to take, on account of the condition in which the case was at the time of treatment. I simply draw attention to that in order to show what strikes me in connection with these reports—that although it may turn out to be a very excellent thing, at present it is not conclusive, and, as Captain Todd himself has said, it will require a very considerable amount of further work in order to establish the true accuracy of strangline.

Mr. Hughes: I should like to mention one fact in regard to this disease. Wherever there is an important horse-breeding district there is an important market town, and there is a possibility of accounting for these sporadic outbreaks—or which some people regard as sporadic—occurring in the district. In our towns we have hotels which have accommodation for from 30 to 60 horses, and on market days it is the common practice for farmers to tie two horses together in one stall. It is quite conceivable, therefore, that when a horse is recovering from a milder form of attack, or even during convalescence, he may be capable of soiling the horse with which he is tied up. I have repeatedly been able to trace the source of infection in that way, and I would direct my friend Mr. Ainsworth Wilson's attention to the possibility of tracing the source of sporadic cases in that way. Hitherto I have regarded the respiratory organs as the chief channel of infection, but I have had numerous instances where the disease has been conveyed by inhalation, for instance, where a foal has been sucking a mare which is affected with strangles. I have seen several cases where the mare has had small abscesses about the udder or towards the perineum; and I have seen the disease conveyed by stallions when they have had nasal discharge. There is one question I should like to ask Captain Todd, because he has made a very exhaustive study of this disease, namely, whether there is greater susceptibility to strangles in the progeny of sires and dams that are known to be affected with the disease than in the progeny of sires and dams that are free from the disease?

Mr. HENRY GRAY: I quite agree with the views expressed by Sir John M'Fadyean as to the etiology of so-called strangles in the horse, and I also agree with the views expressed by Major-General Smith as to the connection of pneumonia and pleurisy and strangles. I will go further than Major-General Smith, and say that if we had no disease known as catarrhal fever or strangles we should have very little pneumonia, no purpura and very few nervous diseases in the horse. To my mind, strangles in the horse is a parallel disease to distemper in the dog. If there was no primary distemper in the dog there would be no pneumonia or no chorea in the dog, and we should have very few cases of enteritis. I have given long attention to the manifestations of these two diseases, and I must come to the conclusion that the symptoms produced by the primary infection may pass off and the animal appear apparently well for some time, or for a long time even, before any secondary symptoms show themselves, such as pneumonia or chorea. I have seen chorea in the dog developed three months after distemper, and in one case eight months after distemper, and I have never seen a case of chorea occur in the dog without distemper. My observations on catarrhal fever in the horse have shown me, in many instances, where the symptoms have abated and the animal has appeared apparently well for a time, that if the temperature is taken for some time after apparent recovery the first indication of a secondary affection will be a rise of temperature. Only last year I had an outbreak of so-called influenza in a stable. All the animals apparently recovered, when one day I was sent for to see an animal suffering from nervous symptoms. I treated him, and he seemed all right for a few days, but eventually he developed pneumonia and the symptoms set in again, and he died. When I made a post mortem examination I found the lungs extensively involved; they were consolidated. Another horse which had apparently recovered fell down in front of my eyes. That developed pleurisy, and also abscesses where the harness went. Another horse developed pleurisy, and I am glad to say both those cases recovered from pleurisy by tapping. I tapped them daily for three days, first one side and then the other. Something has been said about the age at which animals are predisposed to strangles, but I do not believe it is a question of predisposition at all; it is the age at which animals come in contact with infection. We find with old animals coming from the country which have not been exposed to infection, that when they come into London stables they develop the catarrhal fever and end up with pneumonia.

As to the way in which infection is conveyed to the animal, I do not think that matters in the slightest to us as practical men. It is our duty to prevent the disease, but if we prevent the disease I am afraid half our work will be gone. Sir John M'Fadyean mentioned about tuberculosis being conveyed principally by the respiratory tract. I was talking some time ago to Dr. Pollock, who was connected for fifty years with the Brompton Hospital. He told me that out of the hundreds of nurses, washers, and other people about the Institution, some of them having been there for thirty years, he could not remember six people suffering from tuberculosis. If tuberculosis is conveyed by the atmosphere it ought to be a commoner complaint than it is. We must not put experimental evidence against natural evidence. What I want to try and find out is, how does infection take place in Nature? The methods that are used in experiments are not the methods Nature uses, and therefore I think experimental investigations very often mislead practitioners. After all the most important thing is not how infection is conveyed to the body, but how to prevent it.

Prof. JOHN McCALL: In the first place I should like to congratulate Captain Todd on the very excellent and interesting paper which he has given us, and also for the commendable amount of originality and research which he has shown. Practically I am in agreement with the essayist on a great many points, more especially with regard to the predisposing causes of this disease. Debility, colds, catarrhs and over-work are very often associated in my experience with outbreaks of strangles. I think these are most important factors in its production. With regard to the methods of infection, I cannot agree with the essayist when he says that ingestion is the chief channel of infection: I should think that if it were we would get lesions of the mesenteric gland. Also it must be remembered that there are many cases of irregular strangles in which the only symptoms are a catarrh or a cold: that, I think, helps to show that the method of infection is by means of the respiratory passages, and therefore in a typical case of the disease we find we get abscesses formed in the pharyngeal region. I should think that the channel by which the virus is spread was from the mucous membrane into the lymphatics, and so down to the lymphatic glands. I consider that the evidence is more in support of the inhalation theory than in support of the ingestion theory. I have seen outbreaks of this disease among animals going to grass, where the chance of ingestion, although it did exist, was much less than if the animals had been confined in their stable, using water from the same pail or from the common water trough. There is just one other point I should like to mention, although I feel somewhat dubious about doing so in the presence of Sir John M'Fadyean, who has already dealt with the bacteriology of this disease. I waited to hear Sir John give an expression as to the exact classification or distinguishing characteristics of the streptococcus which is said to give rise to the disease, and I noticed that Sir John did not commit himself. The theory has been held by a great many veterinary surgeons that the organism which gives rise to strangles is simply one of the ordinary ubiquitous pyogenic organisms—the streptococcus pyogenes—but other authorities say that the particular one which gives rise to strangles is distinct from this. When we come to regard the scientific evidence on this point we are disappointed. Microscopically, culturally, and in other methods, they are I believe at the present time indistinguishable. So that if we believe in the theory that the organism is simply a modification or an increased virulency of the ordinary pyogenic organism, that explains the occurrence of many outbreaks in which we are unable to trace an infection. I have seen cases of infection amongst foals in a paddock where there have been no cases previously which could not be traced, and I have seen cases occurring on farms where there had been no cases, or at least cases hitherto unrecognised. I think with these facts before us it makes us hesitate before we conclude definitely that there is a distinct organism responsible, and that infection from antecedent cases is always necessary. I do not for a moment wish to suggest that this is a disease which can originate sporadically. Any person with the most elementary ideas of bacteriology cannot accept the origination of bacterial diseases *de novo*. But what I mean is this: is it not possible that the theory that the organism is simply an increased virulency of the ordinary pyogenic organism is true?

Mr. Samson: My bacterial knowledge is not great. As far as the infection of strangles goes I know very little about streptococci or anything of that kind, but there is no doubt that climatic changes will influence the course of the disease. It is not to be said that because you cannot trace infection, the infection has not been there. Take Col. Steele's case, where one lot of horses contracted the disease because they were on the high ground and the others did not have it because they were on the high ground.

Col. Steele: No, on the contrary.

Mr. Samson: Well, it may be *vice versa*. My point is this: you cannot be sure with regard to those horses which did contract the disease that the boxes they travelled in were clean.

Col. Steele: All the horses travelled in the same boxes, mixed up all together.

Mr. Samson: In the same stalls coming across in the ship?

Col. Steele: Yes.

Mr. Samson: With the same attendants?

Col. Steele: No.

The President: You must discuss the paper and not discuss the remarks. We do not want to have a subsidiary discussion conducted by two persons.

Mr. Samson: In Col. Steele's case it has not been proved to me that these horses did not become infected through contagion on the ship or by some of the attendants, or else in the railway boxes. My experience of boxes is that some of them are very filthy. I quite believe the case which Mr. Hughes mentioned. It is a very common occurrence for disease to follow in the studs of farmers who go to market. On market days horses are put in the coach house or anywhere, and the place is very seldom cleaned. I look upon strangles as being very similar to mumps and measles in children, and to distemper in the dog. I am rather inclined to think that each and every horse has to have strangles, the same as every dog has to have distemper. Some horses have it very severely and others do not. The best time of life for a horse to have strangles is when it is a yearling, and if I had strangles break out in my stable in two or three yearlings I would follow the good old Norfolk custom of putting them in the field and letting them roam about. They would get over the disease more quickly by that means than by any other.

Mr. Wallis Hoare: There is one point with regard to the mode of infection of strangles which I think the essayist has overlooked, and that is infection by wounds, either accidental or operative. I have seen a large number of cases of strangles occurring after castration. Of course it might be said the colts had the infection of strangles in them before they were operated on, or that they got infected in the ordinary way after operation; but when you have an abscess forming at the operation wound, when you have the symptoms of strangles appearing after this, I take it that the infection has occurred at the seat of operation. I stand open to correction. The next point that I would draw attention to is, what is strangles, because after reading the paper through very carefully, and after hearing the discussion to-day, I think if anybody was asked to define strangles he would have a great deal of difficulty in doing so. We are told it is catarrhal fever, or sore throat, or anything due to the streptococci. I was always under the impression that there were streptococci in ordinary catarrh or laryngitis. How are we to differentiate them? The only possible way is to put the infected horse in contact with a healthy horse. If the healthy horse does not get strangles you may take it for granted it is catarrh; if it developes strangles then the original horse had strangles.

With regard to treatment, I see citric acid is mentioned as a useful agent. That I believe is the view of Captain Webb. It would take a great deal to persuade me that one drachm of citric acid three times a day could have any effect whatever on strangles or any other disease of the horse. Another point on which I must differ from the essayist is with regard to adult horses. The essayist, I believe, is of opinion that well defined strangles is not common in adult horses. I have seen a very large number of adult hunters with strangles, and my experience has been that if you introduce one case of strangles into a stable, every horse in that stable will develop typical strangles. I have noticed also that if you have a series of typical influenza cases and, by some means or other, you introduce into that stable one case of strangles, nearly every case of influenza which develops after that develops strangles. I have great pleasure in congratulating Captain Todd on a most interesting and instructive paper on a subject about which we know little or nothing, and I hope his investigations will prove satisfactory, and that he will be able to discover an effectual preventive as well as a curative agent.

Mr. NORRIS: I should like to ask one question. Sir John M'Fadyean in opening the discussion seemed to be rather sceptical as to the streptococcus being the cause of the disease. In reading through the paper I see that Captain Todd says on page 96: "Although it is within the bounds of possibility for the streptococcus alone to cause the disease to develop in susceptible animals, clinical and experimental evidence point to an additional factor being necessary in the majority of cases." He goes on then to quote some suggestions which have been made. Is Captain Todd aware of any experiments being made to test whether there is an ultra visible virus present in the case of strangles or not, and if not, does he think such experiments would be highly important?

Mr. HOWARD: As coming from a breeding country I have had some considerable experience of strangles in horses. I am rather surprised that Captain Todd, with his extensive army experience, should seem to think that there is little fear of infection by the respiratory tract. I think there is very ample evidence that a very large percentage of the infection must take place by the respiratory tract. I might relate to you what is well known in the particular part of the country that I live in as regards the spread of strangles. For some years past there has been a depot established for the purchase and shipping of pigs for a particular town. During those years it has become the practice for farmers to drive a considerable distance with the pigs to this depot, and it is well recognised that almost every young horse driven to that particular centre develops strangles. They are not put up in the stable, but they are simply put in the yard where the pigs are deposited. They do not have any opportunity of getting any contaminated fodder at the place, because the rule is that the farmers bring the fodder with them. It is fairly well recognised that within a very short period afterwards those young horses will develop strangles. I think that is fairly good evidence that the majority of those horses must be infected otherwise than by the digestive tract. I notice in the paper that Captain Todd mentions something about those cases in which it has been known that mares have become affected in the genital organs simply after having been tried by stallions. I think he must mean "served," because if they have only been tried by the stallion that is another point which shows that the disease may be spread in a simple fashion, perhaps by inhalation. I should like also to know from Captain Todd or General Smith as regards their experiments in the serum treatment. Have they any idea at all as to the number of horses? because a great number of the horses that they experimented with must already have had an attack of strangles before they ever got them. That is a thing which will have to be taken into account in any statistics, because it is very generally accepted that a horse having had one attack is not so likely to have a second attack, though I have seen saveral horses which have had a second attack. I notice that in the treatment, Captain Todd seems to think that when horses are kept in stalls it might prevent the spread if the practice were adopted of not moving the litter from the stalls. I think he would not find that of much help, as the keeping of the litter there would only make the stall a good deal more unhealthy than if the litter were cleared out. I have seen very good results from a very simple method which has been practised extensively by a few people in my country who get together a lot of young horses at times. They wash the horse's face and nostrils daily with a fairly strong solution of Jeyes' fluid, continuing this for a long time after arrival. I am almost positive that that has been the means of preventing an outbreak of strangles amongst many of those young horses. I am glad to hear from Captain Todd and General Smith that it is possible that the serum treatment may have such a very material effect in preventing the number of roarers, or horses wrong in their wind, after strangles, but I think myself it is possible there is too much stress laid on this question of the number of horses that go wrong in their wind. I think when a horse is wrong in his wind after strangles that in the majority of cases it is only a temporary matter, and that they very often, where they are otherwise sound and healthy, become quite well again. I cannot agree with my friend Mr. Hoare when he says that if a case of strangles is introduced into a stable you are very likely to get every horse in that stable suffering from strangles.

Mr. HOARE: I never said that. I did not say they would become infected of necessity; I said they probably would.

Mr. HOWARD: I may say that I have seen many cases in which a single case of strangles has been introduced into a stable and not a great number of the horses have developed strangles afterwards.

Reply.

Captain Todd: In the first place I would like to thank you for the interest you have taken in this paper, and also for the courteous way you have discussed it. I should also like to take this opportunity of thanking Sir John M'Fadyean for the kind way in which he has received my humble efforts, and also General Smith and others who have referred to the work I have done in connection with the paper. In General Smith we have an officer who is always ready to help in any special work that is for the good of the Service, and he is ably supported by the officers of the Aldershot Command, who have helped me to bring out several details in connection with the paper.

Several points have been raised in connection with the paper, and I will deal with them one by one. First with regard to the method of infection. There is considerable difference of opinion about inhalation or ingestion being the more common method of the spread of the disease. My own opinion is that if inhalation is a very common method of spreading the disease you would see it rapidly going through a batch of remounts in a stable, or through a stud of horses; but it does not go rapidly throngh a batch of remounts. You will find a case crop up one day, then another in four or five days afterwards, and another four or five days after that. I am inclined to think that if inhalation plays an important part it would spread much more rapidly through a stable and more freely from neighbour to neighbour, and that is not my experience of the disease. Then, again, with regard to inhalation, the dust would be almost infinitesimal. With some observations I made with cultures and with pus—putting it on sterile pieces of wood and putting them in various favourable and unfavourable places—it was with the greatest difficulty that I could get it to grow on its most favoured media, and from that I concluded—at least I thought it supported my contention—that inhalation play a very small part in the spread of the disease. General Smith's remarks regarding ingestion, putting cultures or pus with the food in testing the value of serum, I think also supports me.

Someone referred to the mesenteric gland being infected, and said that if ingestion was a common channel of infection abscesses would be found in the mesenteric gland. As is known, in an ordinary case of the disease the discharge from the nostrils, and very often pus from the abscesses, fall on the food, the horse eats it with his food, but you very seldom get mesenteric abscesses after. I do not think it follows at all that because you do not get abscesses in the mesenteric gland ingestion is not a method of spreading the disease. Mr. Hoare referred to wounds as being a channel of infection. I have had no experience, but any observations I have made would not support Mr. Hoare in that respect. Then, again, I have mentioned the small number of cases they had at Melton Mowbray, where the bucket system of watering is in vogue, and I think that also supports me in that respect. There the horses are in boxes standing with their heads out of doors. They can see each other, but they do not drink the same water; their buckets are kept in the boxes, and they do not get strangles. Compared with any other depot with a large number of young horses, those results are most favourable. Mr. Wilson asked a question about the time an animal could be infective after recovery. There is nothing definite known about that. Col. Blenkinsop's South African experience would point to three weeks or a month being sufficient time to keep an animal isolated after recovery in order to prevent the spread of the disease to other units.

Col. Steele referred to exposure, and I think he agrees with me instead of differing. It is a common experience that horses which are in well ventilated stables very seldom get the disease. I do not think the horses that were referred to were exposed to bad weather. By exposure to bad weather I mean standing out of doors, being fed irregularly, and so on, so that the vitality of the animal is so lowered that it would fall a victim to any prevailing disease, influenza, pneumonia or strangles. General Smith said he could not agree with me about fatigue, hard work, and cold. I cannot change my mind about that. I certainly think that we get more strangles during the bad months of the year, February and March, and the chart I gave you of the Continental Remount Depot I think supports this view. Sir John M'Fadyean said that he thought the upkeep of the disease was due to fresh remounts being brought in, but you will notice that in November and December and January there were 200, and only about 18 cases of the disease; it was not until February that a large number became infected. That is long after the period of incubation. If those horses came in December and they did not get the disease till February, I think it follows that the bad weather, scarcity of food, huddling together in sheds, nasal discharge falling about on the food, and so on, favoured the spread of the disease. General Smith also said it was not his experience that animals had repeated attacks of the disease. Captain Clive Webb informs me that it is quite a common thing at the Remount Depots in India for foals—in India they do not get their horses as three and four-year-olds; they are bred in the district and come their at various ages, from six months to one year—to get repeated attacks of disease. The authorities out their have a system of labelling the horses with a tin label, showing the disease, and it is a very easy thing to know whether an animal has had an attack of strangles before or not.

With regard to the advisibility of protecting horses, it has been brought out in the discussion what an enormous time is lost from horses losing their training. When you consider that quite 25 per cent. of horses go wrong in their wind after recovering from strangles—I refer now to horses that join regiments at four or five years of age—I think it is well worth while to try and prevent that loss, which is a very serious monetary loss. A question was raised by Mr. Hughes as to heredity. I cannot enlighten him about the pedigrees of horses in the Service; they are bought with pedigrees unknown, at four and five years of age. All I know is they are sound when they come into the Service; they are examined very carefully by the veterinary officers attached to the Remount Depots. Lately in Aldershot we have been examining them after recovery from strangles, and we find that it is by far and away the most common experience that they become roarers: and it is also interesting to know that those who suffer from catarrhal fever without abcess formation—which in reality are the atypical forms of strangles I have referred to—are just as liable to go wrong in the wind as those who have the abscess formation. In connection with roaring I would like to state that the examinations, the results of which I have given you, were made by independent officers attached to cavalry regiments in the Aldershot Command. They were as much interested as myself in the results, and were very careful in making these examinations. I think the results are most encouraging, and I hope that they will be taken up by the members of the Society, who I trust will take every opportunity of verifying or refuting them. Mr. Norris asked me if I knew of any experiments that had been made to test whether the disease was caused by catarrhal virus. I know of no experiments which have been made in that direction. My own opinion is that the streptococcus equi, plus the predisposing causes I have referred to, is sufficient to cause the disease.

Mr. Hoare referred to the difference between sore throat and the different forms of disease, and asked what was strangles, and what was not strangles. It is very difficult to say in a case of catarrh whether it is strangles or not, but I think one is wise, and is working in the interests of the client, if one treats such a case as strangles, and points out the danger of allowing the horse to mix with others. That is all one can do. Mr. Hoare also raised a point in connection with the citric acid treatment which was tried by Captain Webb. Captain Webb used to perform tracheotomy in severe forms of laryngeal affections of the disease where the breathing was difficult, and he had no luck at all. When tracheotomy was performed most of the cases died. But after using the citric acid treatment he found that the breathing was relieved in two or three days, and that there was no need to perform tracheotomy. Mr. Hoare said something about a young horse suffering from strangles being brought into a stable full of hunters, and the old horses getting it as well as the young ones. That is certainly not our experience in the army. It is a common thing for a young horse to get strangles in a stable full of old horses, but that would be the only case that we would get in the stable. One would certainly take the young horse away and isolate him, but we do not find the disease spreading to the old horses. I can bear out Mr. Hoare when he says that if you get an outkreak of influenza and then introduce a case of strangles, most of the other horses will get strangles. I think that is caused by the vitality of the animals being lowered by the influenza, thus making them victims of strangles. With regard to the point raised by Mr. Howard about mares being tried, the case that brought that to my mind was that of a mare which was tried by a horse which was recovering from strangles. When horses try mares they rub their noses close up against the vulva, and I think that was the channel of infection in that case.

Mr. Howard also said it was important to know whether serumised horses had had strangles or not. The chances are that some of them had recovered from strangles. On the other hand there were a certain number of controls used in Woolwich that were referred to by General Smith, and the results from serum inoculations were very much better than they were in the controls, so that supports the evidence as to the value of serum in the prevention or the modification of the disease when horses get it. With regard to the litter in the stalls, as I pointed out in my paper, the danger in this connection is more from eating the fodder than from lying on it. Quite recently I had a case of some bedding by accident being removed from a case of strangles into another box. A young horse ate the bedding and developed the disease. With regard to the connection between pneumonia and strangles, my experience has not been that of General Smith. Those outbreaks of pneumonia I have had to deal with have been mostly of the form known as contagions pneumonia, and I can only call to mind about two cases where I had a double infection—strangles and pneumonia—to deal with in one horse. I rather look upon the two as separate diseases.

With regard to Strangline, I would like to state that the cases I brought forward are more in the form of a preliminary note than anything else, and to show that after all there is some value in this new method of treatment. Sir John M'Fadyean seemed to think that I could not attribute my success to the Strangline because the change in the patient took place so soon afterwards, but if you look at case 4 you will find that the animal had been ill from January 14th to February 10th, which is a very long time to have a patient under your care, and it is about time one thought of doing something. You will notice that very soon after February 10th that animal got better, I have not given the date when it was noticed to be getting better. As a rule the improvement was to be noticed on the third, fourth or fifth day. I know there is a negative phase following on the introduction of any vaccine into human beings or animals. That negative phase was not noticed in my observations. The animals did not go off their feed or show any signs of constitutional disturbance, so I conclude the negative phase was a very small and short one. With regard to case No. 7, the life of that horse was undoubtedly saved by the use of Strangline. It had been suffering from December 6th to January 30th, which is a very long time, and the improvement was most marked after five days. I agree with Sir John when he says that a large number of observations would have to be made before we could give value to this agent. In other diseases treated by vaccine there have been bad results as well as good results. It may be that there are stages of the disease that are unfavourable for treatment by vaccines. I am inclined to think that the acute stages of the disease would not lend themselves to treatment by this method, but I certainly do think that we are on the track of a very valuable remedy for the treatment of these irregular and protracted forms of disease. I would like to thank you for the very patient hearing you have given me.

[Adjourned for luncheon.]

PREFACE.

I feel an apology and explanation are due to the members of the Association because, after having arranged to write an original paper on Rickets, I am presenting one that does not fulfil these conditions. I had left the formulation of my notes and the remainder of the work to be done during my annual spring holiday. This was rendered impossible by my being called away to South Africa at a few days' notice to the death bed of a great friend. Immediately on my return I was too unwell to undertake any writing. Following this my partner's holiday transpired, and still further rendered the question of writing impracticable. My friend, Dr. Findlay, with whom I am now doing experimental work on this subject, has very generously given me full permission to use his very ably written pamphlet. I am availing myself of this for two reasons. First, that the Association may have the opportunity of discussing the subject according to arrangement. Secondly, because Dr. Findlay's views and my own, on the subject of Rickets, are identical.

I should like here to emphasise the point that, while the Medical profession is only now beginning to accept the views as contained in this paper, many members of the Veterinary profession have, I believe, held them for a number of years.

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THE CAUSATION OF RICKETS.

Many and varied have been the causes assigned for rickets. Almost every prejudicial circumstance in the life of the infant—and in that of the parent too, for that matter—has been cited as the etiological factor.

Etiological Theories.

According to Kassowitz and some of the German school, it is of congenital origin, a theory, however, mainly founded, at least by its earliest adherents, on macroscopic appearances alone, and not on the finding of any characteristic histological changes in the bones. In every infant the bones are soft, the abdomen may be protuberant, the fontanelles will be open, and there will be some swelling of the costochrondal junctions, and yet it was on the ground of these facts alone that such a theory was based. The later advocates of this theory, however, did examine the minute histological changes in the bones, but as much of the material studied was obtained from stillborn children, and as the pathological anatomy of osteochondritis syphilitica and rachitis was at that time only imperfectly understood, these two conditions were much confused, and it is not improbable, therefore, that many of the supposed rachitic lesions were in reality of a syphilitic nature. As these two processes came to be differentiated from one another, each succeeding published record of work done on this subject showed a marked diminution in the proportion of congenital rickets. Kassowitz in 1882 considered that 80 per cent. of all children born were rachitic: in 1897 Tschistowitsch put the proportion at 12 per cent., while more recently (1902) Escher, after investigating a large series of cases both clinically and histologically, was only able in one single instance to make a diagnosis of congenital rickets.

There is, of course, such a condition known and described as "fœtal rickets," but it is considered by the majority of observers an entirely different disease from that which ensues after birth, and is, moreover, as rare as ordinary rickets is common. It will probably be better then, at least for the present, to classify it apart, and designate it by one of its pseudonyms—for example, "osteogenesis imperfecta."

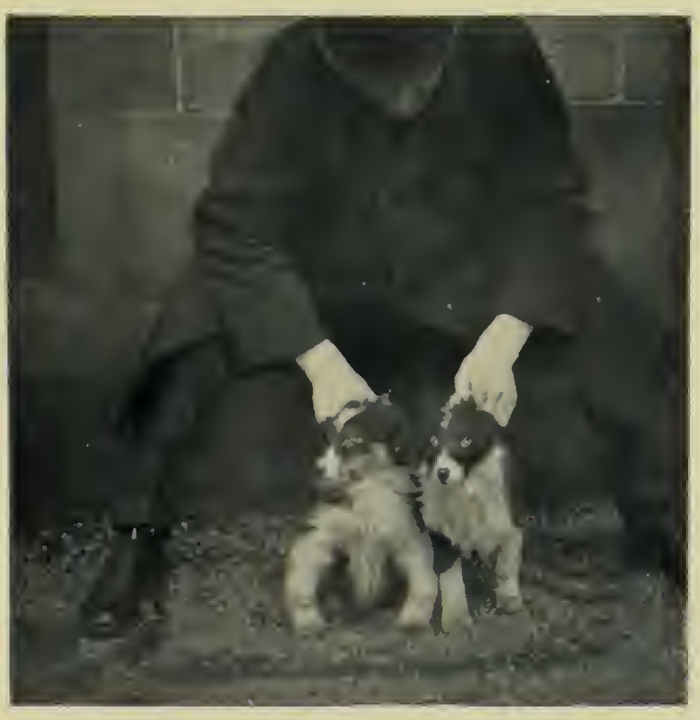
Closely allied to the above theory (congenital origin of rickets) is that mainly advocated by Siegert, who holds that the condition is hereditary, and dependent on some inherited weakness or predisposition. This, after all, is merely a case of begging the question and failing to discuss the main points at issue. Hausen, however, reports the case of a stallion who begot seven rachitic foals. Later, two of the mares, one of which had given birth to three and the other to two of these rachitic foals, became pregnant to another stallion, and bore healthy non-rachitic offspring.

It is during extrauterine life, however, that most present day authorities believe the etiological factors play their part, and the general consensus of opinion would ascribe the disease to incorrect feeding, bad hygiene, and want of sunlight. Of all these possible malign influences it is some error in feeding which receives most credence and support. By some it is said to be due to an entire want of suckling, to the mother not nursing the child long enough, to suckling at too frequent intervals, and by others again to prolonged lactation. Siegert found that out of 845 artificially fed infants 81 per cent. developed rickets, while in 923 infants who had been nourished at the breast for at least four-and-a-half months, only 31½ per cent. became rachitic. On the other hand, Holt mentions that although negro and Italian children in New York are entirely breast-fed, they are exceedingly prone to develop the disease.

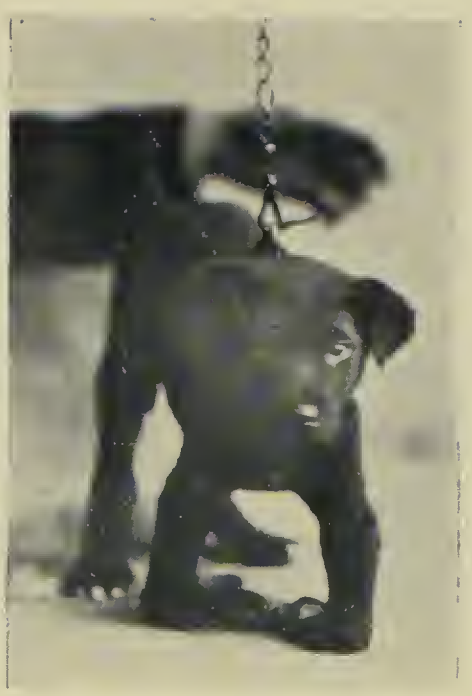
In artifically fed infants and in the feeding of children, almost every possible quality and combination of the different constituents have been credited with the power of inducing the disease. Some say it is consequent on a deficiency of proteid, others on an excess of carbohydrate, but perhaps the majority consider that it is due to a deficiency of fat and proteid with an excess of carbohydrate. This is a combination of factors which, it must be remembered, may be present in the food of both naturally and artificially fed infants. According to Holt and Cheadle it is in the presence of a low percentage of both fat and proteid that rickets is liable to result, while Bland-Sutton teaches us that it is entirely due to a deficiency of fat. The latter's well-known experiments in the London Zoological Gardens with the lion cubs and young monkeys lend great support to his view. By only one change in the general régime of these animals, namely, the addition of fat and cod-liver oil to their diet, lion cubs were reared free of rickets for the first time in ten years. Ashby, while lending his support to the idea of a low percentage of fat and proteid, considers that it is not improbable that fermentation of the carbohydrates may produce toxins, which are responsible for at least some of the phenomena of the disease. Heitzman (11) considers that "lactic acid" is the baneful product, and claims to have induced the condition experimentally by its administration. He believes that the lactic acid results from the fermentation of the carbohydrate and irritates the ossifying tissue; but other observers have not been able to confirm this formation of lactic acid, and have failed to bring about rachitic changes by its administration.

Esser (12), who has lately written on the subject. believes that the disease is due to over-feeding, and bases his theory mainly on the similarity of the blood pictures in rickets and chronic over-feeding. He contends that over-feeding produces a chronic gastro-enteritis, and results in a deficient absorption of the food constituents. Cheadle (13) too, in one place speaks of rickets ensuing on starvation, which is, however, contrary to clinical experience.

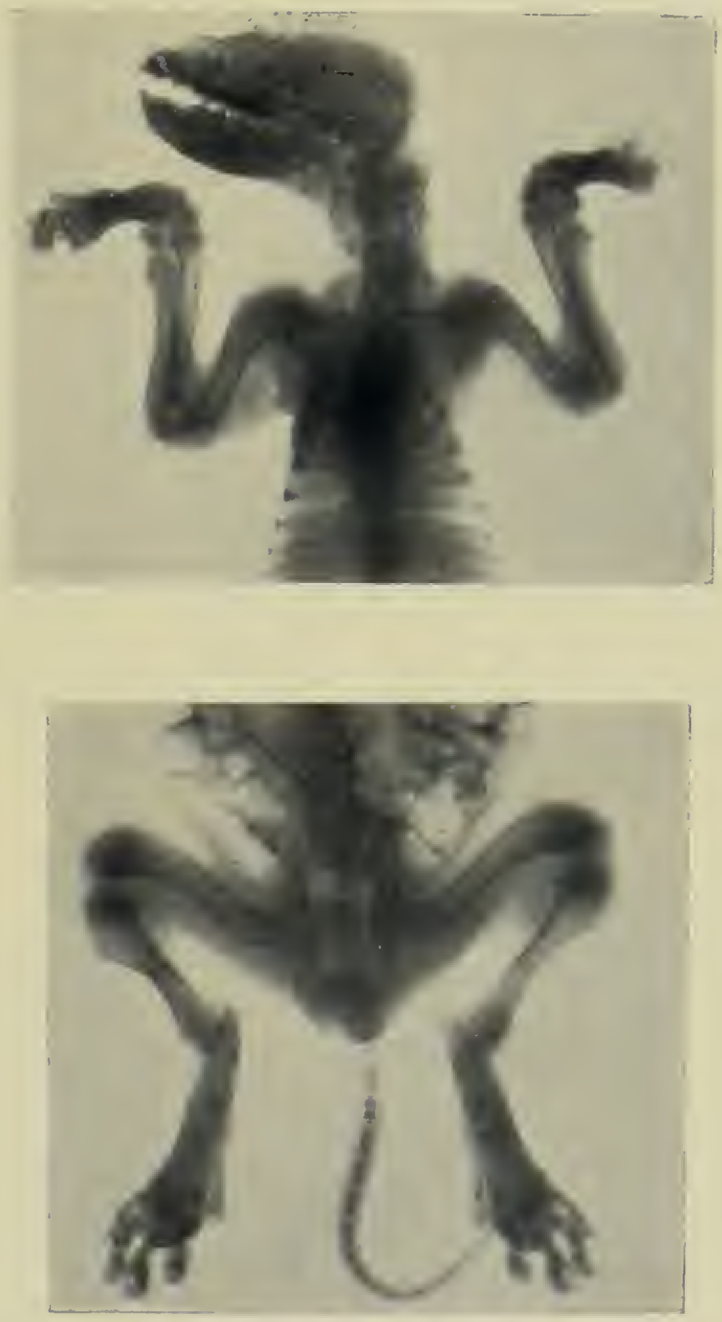
Considering the fact that the rachitic changes in the bones are in great part characterized by deficient calcification, it is not surprising that some observers have looked for the etiological factor in a defect of lime metabolism. It has been attributed to an absence or deficiency of lime in the food, to a defective anabolism of the lime salts, and, lastly, to an increased katabolism of these same salts—in short, to all the possible variations in the process of calcification. Chossat (9) produced curvature of the bones experimentally by depriving animals of earthy salts, but Friedleben doubted the condition being true rickets. Voit (14) also believes that he was successful in causing rickets by depriving animals of calcium. Katz (15) saw typical rickets in a fowl which had been confined in a narrow cage for some months, and which he considered due to the fact that the bird could not get a sufficient amount of lime. Reimers and Boye (16) also noted some changes in the bones of dogs fed on a diet poor in lime, but very different from that they observed in cases of spontaneous rickets. The bones in the experimental animals were soft, with some thickening of the epiphyses. The bony trabeculae were merely thinned and did not reveal the presence of any osteoid tissue. In cases of spontaneous rickets, however, the epiphyses were enormously thickened and much osteoid tissue was present. That it is due to a want of lime is unlikely when one considers that farinaceous foods and cow's milk—the diet on which rachitis is supposed to flourish—contain more than an abun-



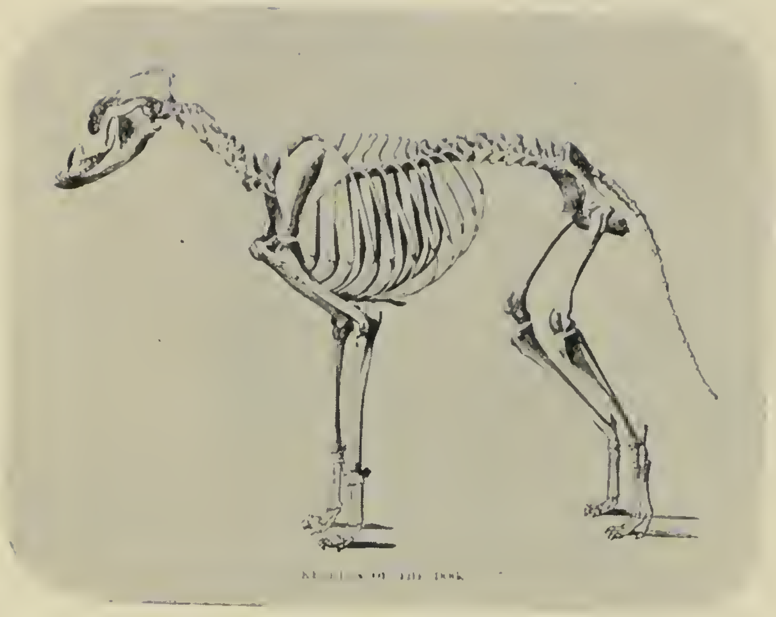
The two collie puppies, ten days before death. Note the extreme degree of bending of the fore legs in the rachitic animal.



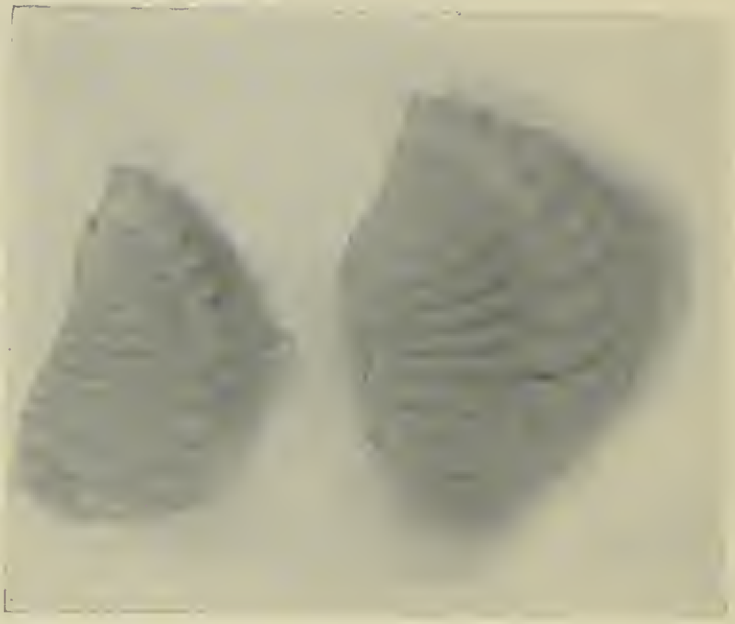
Rachitic Great Dane three months puppy.
(Skeletons on following page)



Radiographs of Skeleton showing Rickets.
Great Dane pup, three months.

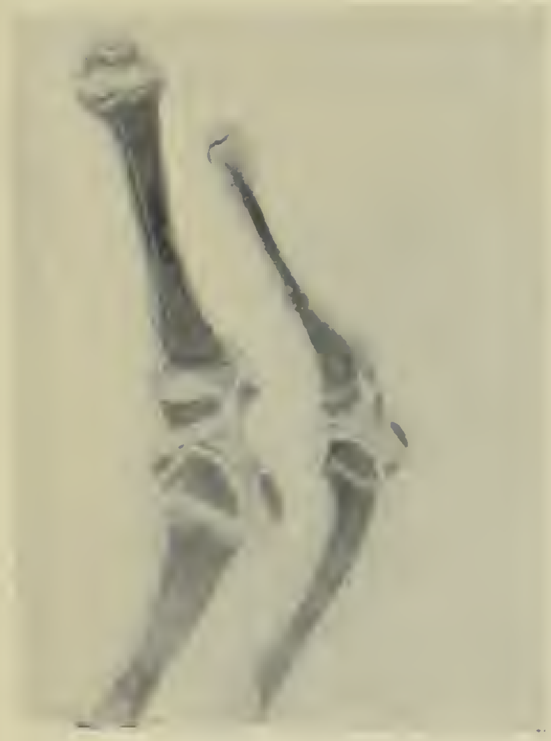


Skeleton of dog
(normal)



Inner aspect of the thoracic walls in healthy animal,

Rachitic animal shewing the marked rosary or nodules at the junction of the ribs and sternum.

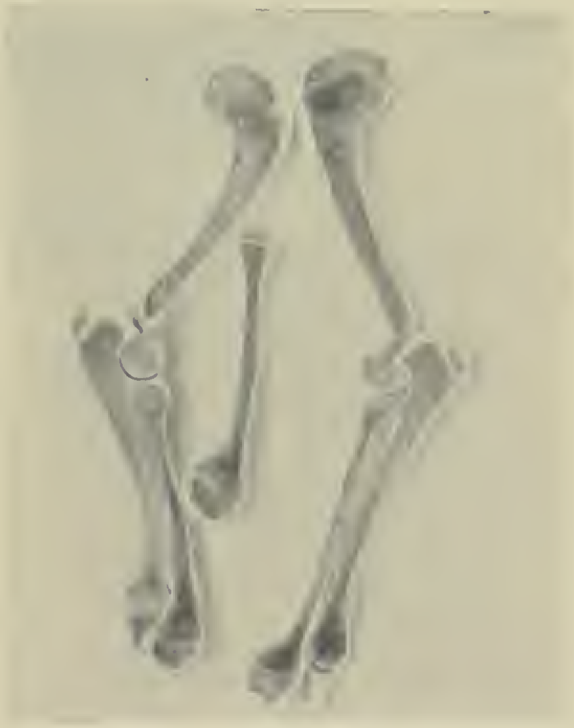


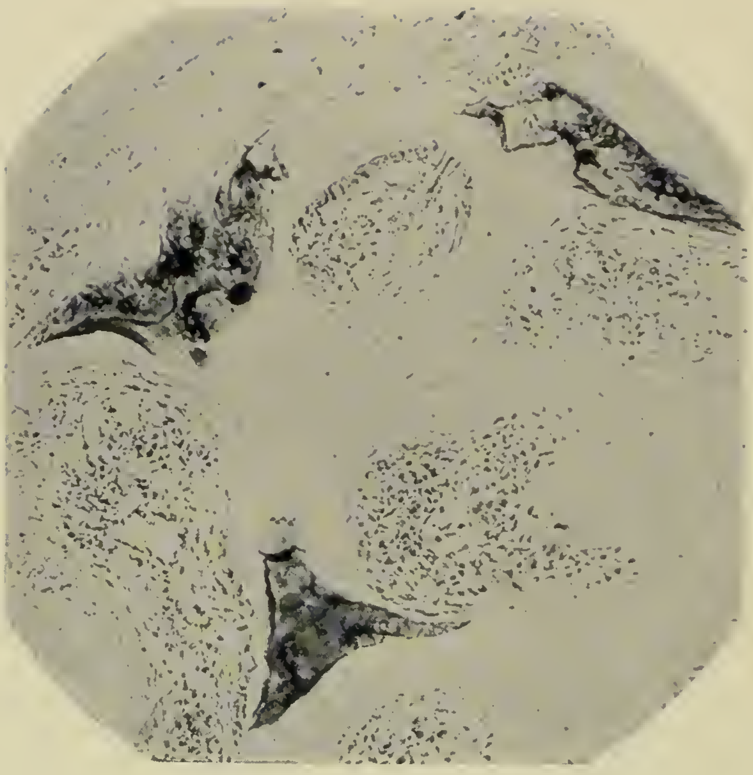
Longitudinal sections of bones of hind legs from healthy animal showing normal conditions.

From rachitic animal showing the thinning of shafts, and great irregularity and widening of epiphyseal lines.

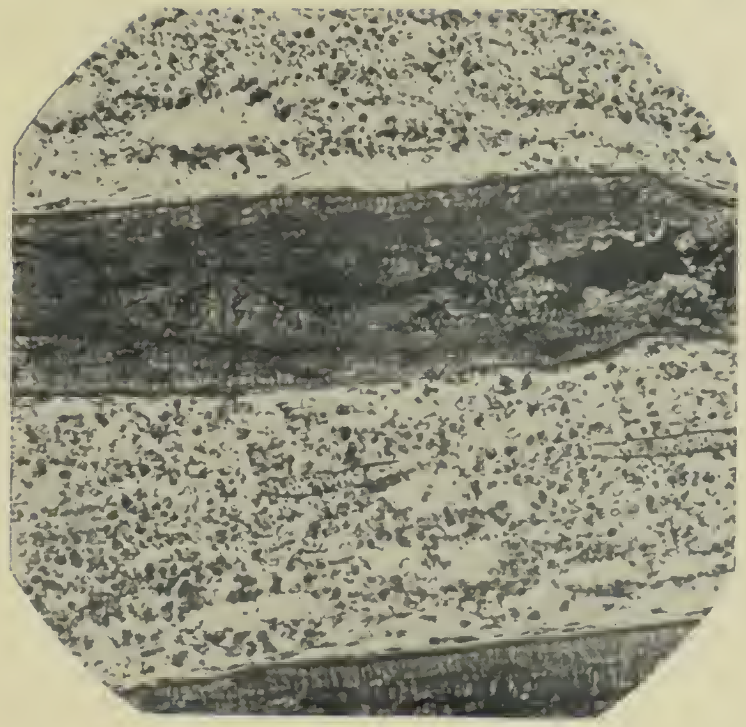
Sections of bones of forelegs of the two collie puppies. Healthy puppy.

Rachitic puppy: Note the bending of the humerus, and the great thickening of the epiphyseal lines in the radius and ulna of the rachitic puppy.

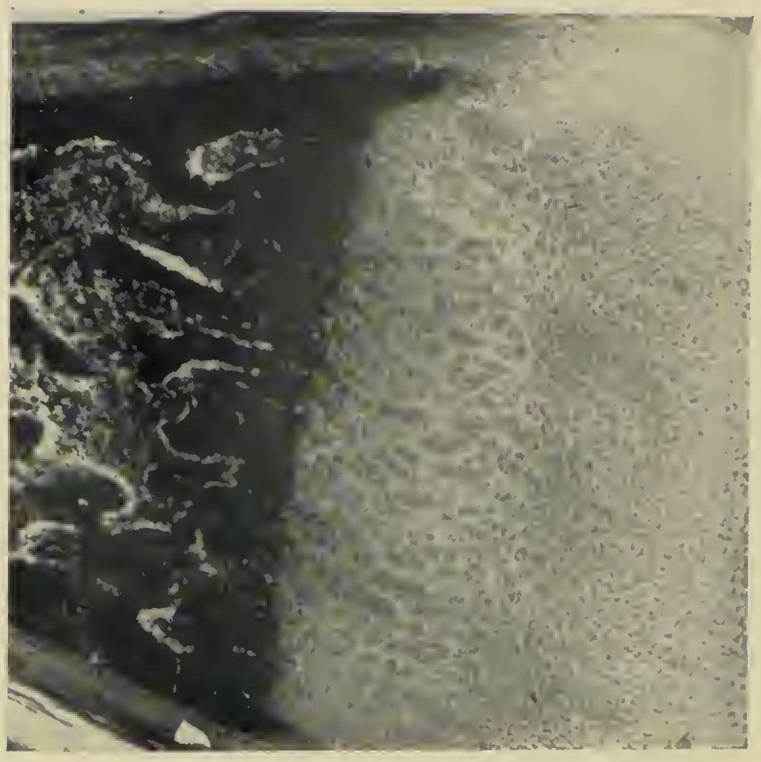




Photomicrograph of a section of rachitic bone stained with thionin blue as advised by Schmorl for the demonstration of bone cells. Note the presence of much osteoid tissue and the dwarfed processes and irregular arrangement of the bone cells.



Section of ordinary bone for comparison. Here there is no osteoid tissue, The bone cells are regularly arranged, and extend right to the margins of the trabeculae. In one trabecula some unabsorbed fœtal bone is present.



Photomicrograph

dance of that substance. There is no doubt, of course, that during the rachitic processes there is a deficient absorption of lime by the newly-formed bony tissue, and just as truly is there an increased katabolism of the same salts, but it is just on what this perversion of metabolism depends that the crux of the whole question lies.

Hygiene and climate are acknowledged by most authorities to play some part in the etiology of rickets, but only to a slight extent, and in a very general way. To this question, however, I will return later.

The infective theory has been mooted by many, but as yet there is no definite proof in favour of any such idea. Drs. A. Torane and Salvatore Forte (17) believe they have induced the disease in rabbits by inoculating them with watery and alcoholic extracts of the fæces of children suffering from rickets and diarrhœa. Curiously, though rachitic changes are said to follow on injection of either the alcoholic or watery extract, a mixture of the two extracts is quite inactive. Moussu (18) also is of opinion that it is an infectious disease, as he caused a healthy animal to take rickets by confining it in a cage along with a rachitic one; and then, without subsequent disinfection, he confined another healthy animal in the cage, and it also in turn succumbed to the disease. This experimenter, in addition, inoculated various kinds of animals with emulsions of rachitic bone marrow, but without any effect, unless he injected what he calls a very virulent material, and also confined the animals in the above-mentioned, or some other similarly-infected cage.

Syphilis, too, has been considered answerable, and it is possible that it may act as a predisposing factor; but that it is the prime etiological cause, as Parrot (8) and Marfan (19) consider, is a view which has not received much support in this country, and seems, moreover, to be entirely negatived by the fact that rickets can be easily induced in the lower animals.

The Effects of Deprivation of Exercise.

The great diversity of opinion which prevails regarding the etiology of this disease is shown by the above brief account of the various theories, and is sufficient proof that as yet the true etiological factor has not been discovered. Rievel (20), while summing up the recent work on this subject, suggests that rickets may not have the same origin in all cases, and that many factors may play a part in its causation. It is my intention, however, as the result of some experimental work which I have recently carried out, and by a critical survey of the various influences which affect the child, to show that there is one factor, and that a very potent one, in inducing the disease—namely, want of exercise.

Lack of exercise as a factor in the pathology of the disease has received remarkably little attention, though to my mind it is of the utmost importance, and in future will always require to be taken into consideration when discussing experimental rickets. Though confinement is mentioned by several authors, its mode of action is either misunderstood, or it is supposed to work along with some other baneful influence, and to play a subsidiary part. Dudgeon (21) believes that it acts through lack of fresh air and sunlight, while Clement Lucas (22) mentions it as a contributory factor to incorrect feeding, and Esser (12) to overfeeding. By depriving young dogs of exercise I invariably induced the condition, though they received as much fresh air and sunlight as the control animals. Moreover, their kennels were cleaned regularly. It is not an uncommon experience for experimental animals to develop rickets, consequent, I believe, on the confining of the animals in cages, an almost invariable practice in experimental work. In this way we can explain the case of the rachitic fowl reported by Katz, and also the results of Moussu's experiments. It is in a similar fashion that climate exerts its influence. In warm, genial climates the children will be out much during the day, while in temperate and treacherous climates like our own, especially during the winter, children are exceedingly likely to be cooped up in the house for days, and even weeks, at a time.

It was some two years ago, when commencing an investigation on experimental rickets, that my attention was first attracted to this subject. I attempted to produce rickets in puppies by modified feeding, giving them such generally recognised rachitic diets as bread and water, oatmeal and water, and rice and water, and yet not one developed the disease. They invariably wasted, became marasmic, and died, thus confirming the well-known fact that marasmic infants never become rachitic. But the control animals, at least those which did not become atrophic consequent on diarrhœa, though being fed normally—as, for example, on milk and porridge—all became affected.

In the first series of experiments three control animals developed rickets, one of them, however, less severely than the other two. This animal was fed in a similar fashion to the others, and spent most of its time in their company; but, owing to the fact that it was ultimately intended as a companion, it was exercised by its prospective owner once or twice daily. Accordingly it seemed to me not improbable that in the confinement and want of exercise—the only abnormal factor in the régime of these animals—one had to deal with the etiological factor. I allowed the comparatively healthy animal to get still more exercise for about a week, when all appearances of rickets practically disappeared. On once more confining him with his brothers and sisters he again rapidly became affected. Two of this litter suffered so severely that they were unable to walk, and merely shuffled about the floor of the kennel as if they were affected with paraplegia or diplegia. Similar experiments have been carried out on two other sets of animals, with exactly the same result. By subjecting the animals to no other abnormal condition than the want of exercise, marked rachitis has invariably ensued.

The table given comprises my observations in chronological order on sixteen animals. Nine of these were treated by confinement and deprived of exercise, but they were fed normally. One (No. 14) died of marasmus, too early, however, to allow of the development of the disease, but the other eight (Nos. 2, 4, 5, 8, 9, 10, 11 and 15) became rachitic to a marked degree. Two (Nos. 1 and 3) were treated by modified dieting and were allowed exercise. Both died of marasmus, but without showing any signs of rickets, although they lived beyond the age at which the disease usually appeared in the experiments. The other five (Nos. 6, 7, 12, 13 and 16) were used as controls. They were all allowed exercise, but as regards feeding, housing, and hygiene were treated in a similar fashion to those which developed the disease, and they all remained entirely free of any rachitic manifestation.

I will quote in more detail my last series of experiments. I obtained three collie pups, aged two months, from one litter. These three animals were kept in one house, were fed in exactly the same manner on a diet of oatmeal porridge and milk, and were subjected to the same atmospheric conditions as regards amount of sunlight, warmth, and purity of air. Two, however, were confined in a small cage closed on either side but open above, and only covered in front with wire netting, while the other was allowed to run about the room at large and play with a cat. All three at first suffered from diarrhœa, and one of the confined animals in subsequence died, but without showing the slightest evidence of rickets. The other two recovered, and steadily put on flesh, and, as seen in the charts A and B, they increased in weight by almost exactly the same amount each week. The puppy allowed to run about developed along normal lines, while the one confined ultimately became rachitic and presented a very typical picture. Fig. 1, from a photograph, shows the contrast between the two animals. The rachitic pup continued to take his food well, and was not troubled with diarrhœa. The first change noticed was a certain degree of languor. When allowed out of the cage he seldom frolicked about, and he was unable, except with the greatest difficulty, to ascend even a very gradual stair. His legs ultimately became bandy, and the beading of the ribs got more marked. His lethargy increased and he lost strength, and, though almost as large and as heavy as his sister, he was frequently overturned by her simply knocking against him.

Post mortem. The difference between the bones of these two animals was most apparent. In the control the bones were straight and firm, the costo-chondral junctions normal in appearance, and the epiphyseal lines narrow and regular. In the other animal the long bones were curved and exceedingly soft, the epiphyseal lines were broad and irregular (Fig. 2), and there was marked swelling with hyperplasia of the costo-chondral junctions.

In this paper I only intend to deal with the etiology of the condition, and consequently will not devote much space to the pathological changes. In order, however, to show that the condition I had induced experimentally was in reality rickets, it may be advisable to recount the main histological findings in the bones. I contented myself by examining the ribs and long bones. As previously mentioned, the bones were soft and pliable, and in some instances could be flattened between the finger and thumb. This pliancy of the bones was most striking in comparison with the hardness of the bones of normal animals. There was distinct swelling of the epiphyseal ends of the long

TABLE SHOWING RESULTS OF EXPERIMENTS

No.	Sex.	Date when came under observation	Age when came under observation		Treatment	Date when Rickets Developed †	Cause of death	Date of death	Time under observation in weeks
1	M	May 11, 1906	At birth	All of one litter	Dietetic	—	Marasmus	Aug. 5, 1906	12
2*	F	May 11, 1906	,,		Confinement	July 21, 1906	Killed	Aug. 17, 1906	14
3	M	May 11, 1906	,,		Dietetic	—	Marasmus	Aug. 18, 1906	10
4*	M	May 11, 1906	,,		Confinement	July 20, 1906	Killed	Aug. 23, 1906	15
5*	M	May 11, 1906	,,		Modified confinement	July 30, 1906	,,	Aug. 28, 1906	16
6	F	April 11, 1907	4 weeks	From one litter	Control not confined	—	,,	June 18, 1907	10
7	M	April 11, 1907	,,		Control not confined	—	,,	June 17, 1907	10
8*	F	April 11, 1907	5 weeks	From one litter	Confinement	May 14, 1907	,,	June 9, 1907	8½
9*	F	April 11, 1907	,,		,,	May 14, 1907	,,	June 13, 1907	9
10*	F	April 11, 1907	,,		,,	May 14, 1907	Broncho-pneumonia	June 21, 1907	10
11*	M	April 11, 1907	4½ weeks		,,	May 12, 1907	,,	June 9, 1907	8½
12	F	June 3, 1907	8 ,,		Control not confined	—	Killed	July 7, 1907	4½
13	F	June 3, 1907	9 ,,		Control not confined	—	,,	July 7, 1907	4½
14	M	Nov. 30, 1907	8 weeks	From one litter	Confinement	—	Marasmus	Dec. 31, 1907	4½
15*	M	Nov. 30, 1907	,,		,,	Jan. 7, 1908	Killed	Feb. 4, 1908	9
16	F	Nov. 30, 1907	,,		Control not confined	—	,,	Feb. 4, 1908	9

* Animals which became rachitic.

† These dates are of course only approximate, it being impossible to say exactly from clinical appearances alone when the disease commenced, but at these dates the animals were undoubtedly rachitic.

bones and of the costo-chondral junctions (Fig. 3). Section of the bones revealed enormous thickening of the epiphyseal lines, which measured in some instances 12 mm., and great irregularity and increased vascularity of the same (Fig. 4). Microscopic examination showed much hyperplasia and irregularity of the epiphyseal cartilage—deficient calcification at the growing zone with the development of much osteoid tissue (Fig. 5). The osteoid tissue was, however, more cellular than that occurring in human rickets, but this can perhaps be accounted for by the more rapid development of the disease in the experiments. The little bony tissue present was composed of cells with dwarfed processes irregularly arranged, and contrasted markedly with what occurs in normal bone (Fig. 6).

When we come to correlate these experimental findings with the conditions under which rachitic children live, it will be found that there is much to support this theory of the cause of rickets.

Rickets is a disease of the temperate zone, being very rare, and, in fact, practically unknown, in tropical and sub-tropical countries. The staple diet in these climates is anything but nourishing, the people living mainly on rice, or some other similar cereal, exactly the kind of food which is supposed in this country to generate the disease. The mothers in these countries no doubt live under more natural conditions and suckle their infants; and, as is well known, in Japan, where rickets is extremely rare, and also in Italy, the mothers suckle their children for unduly long periods, and yet with no untoward results. The climate permits these races to spend much of their time in the open air, and the houses are merely used as sleeping apartments. It is this living so much in the open air which probably accounts for their immunity. As previously mentioned, negro and Italian children, though not subject to the disease when reared in their native lands, become almost invariably affected when dwellers in a city, as in New York, where they are said to be the worst-housed people in the States. The fact that rickets occurs chiefly during the winter months lends further support to this theory. It is during the spring that the city dispensaries are crowded with rachitic children, as a result of the winter's confinement. During the winter, in such a climate as ours, a mother, in order to do her child justice, must be on the watch for, and take full advaniage of, every dry half-hour during the day—rather an arduous task for even the best disposed of mothers.

It is a disease which is found chiefly among the children of urban populations, being comparatively rare in the country. Further, it is among the children of the poor and the working classes that we find the disease most frequently, and not among those of the rich. One of the main differences between these two classes of children—the rich and the poor—is that the former has usually a nurse to himself, while the latter has to be content with as much or as little spare time as his almost invariably overwrought mother can devote to him. The one is taken out into the fresh air several hours daily, and is entertained at home in an airy nursery, while the other is induced to lie quiet in bed or in some corner of the kitchen, and give as little trouble as possible.

The poor man's child undoubtedly gets out little; his mother may have too many children and has no spare time, or she may not perhaps have the inclination to take her child out for an airing. Or it may be that he is the child of some single or widowed woman, who, in order to earn her livelihood, puts him out to a day nursery, where he may be fed and cleaned regularly, but of exercise and entertainment he he will receive a minimum. Moreover the uneducated are notoriously afraid of fresh air in case their children may catch cold by exposure to the weather, so that, with good intention however, the child may not be out for days or weeks at a time, more especially should he be so unfortunate as to suffer from some bronchial catarrh. Again, in cities the poor live in tenements up many flights of stairs, so that there is every inducement for the mother to stay indoors. Well-to-do people find living in tenements a deterrent to going out—how much more, then, must this mode of living affect the irresponsible poor. When the labouring man's child is taken out for a walk, he is almost invariably tightly bandaged to his mother's side with a large shawl, which must impede the movements of his limbs and deprive him of almost all exercise. The great variation in the incidence of this disease among the poor in the country and in the town may perhaps be accounted for by the very different conditions under which they live. The country poor live in cottages, so that the children can be taken out of doors with a minimum of trouble, the very opposite to that which prevails in the town.

This confinement of the children, which we find wherever rickets prevails, does of course deprive them of some fresh air and sunlight, and thus reduce their resisting powers, but it is not entirely, or even mainly, on these grounds that it exerts its baneful influence, as my experiments prove. It is due to the want of exercise which invariably goes along with, or is consequent on, the confinement. Children confined to the house are much more lethargic than those taken out of doors. They sleep more, are less vigorous, and are content to lie at rest without evincing any desire for the exercise of their limbs. Every observant parent and physician must have noticed this, and in the case of a healthy child, it is possible from this fact alone to say whether or not he has been out for his usual airing.

As regards feeding, much of the evidence is of an exceedingly doubtful nature. I cannot believe that the poor in the town feed their children so much less intelligently than those in the country as to account for the great preponderance of the disease among their offspring. Their children are often overfed, but is that not equally true of the well-to-do town child? Does the rich parent never overfeed his child, and does the well-to-do mother invariably, or even usually, suckle her infant? As is well known, several observers have been unable to induce rickets experimentally by incorrect feeding. Further, is it not during the summer months, when gastro-enteritis is rife and there is consequently a diminished absorption of food, that rickets improves, while in the winter time, with no gastro-enteritis, rickets flourishes? It is questionable, too, if a child, deprived of a sufficient supply of fat and proteid, will steadily put on weight, and yet such is not infrequently observed in both experimental and spontaneous rickets. In all my experiments the animals continued to increase in weight, for some time at least, after the condition had definitely declared itself.

Rickets is admitted on all hands to be an eminently curable disease, its dangers depending on the susceptibility of the children to pulmonary mischief, and on the deformities which result, especially in the female sex. In treating the condition, the physician often pays as much attention to the general hygiene as to the question of feeding, and he may at the same time prescribe cod-liver oil and a haematinic, of either of which, however, it is doubtful if much is absorbed. Every one has had the experience of seeing marked improvement take place in two or three weeks. It may not be possible for these poor people completely to revolutionize the hygienic conditions of their homes, but it is certainly possible to take the child out more often in the open air, to give him a daily bath, and to practise a little massage and passive movement of the limbs. Much more easy is it to my mind, materially to alter the hygiene of the child than the manner of feeding, even to a slight extent. Will a child who has been accustomed for many months to "the run of the house," with all its attendant delicacies, allow much curtailment of its diet, and remain quiet on a simpler and healthier fare? Even should this revolution in feeding take place, will the intestinal mucosa return so speedily to its normal, and allow of perfect absorption? Again there are cases in which it is impossible to detect any fault in the manner of feeding, though the mother will frankly admit not taking the child out in case it might catch cold. Holt, though he holds that rickets is due to incorrect feeding, admits that he cannot find any error in the dieting of the negro and Italian children in New York.

In the majority of cases a child develops rickets before it has learned to walk, that is, when it is still dependent on its mother and nurse for its exercise. There are many cases, however, in which the child has commenced to walk only to go off its feet later and become markedly rachitic. Perhaps in most of these instances the condition is secondary to some illness, as, for example, measles with bronchitis; and, in order to avoid a recurrence of the complication, the child has been kept indoors, and guarded against the slightest draught of fresh air. But there are other cases in which there is no such history of an intercurrent illness, but owing to the mother being pregnant, or ill from some other cause, the child has not been taken out sufficiently often. In neither of these classes of cases can we call to our aid as a cause the manner of feeding: for months the feeding has been the same, and in many instances such as we cannot take the slightest exception to. In all, however, we get the same story of confinement, and the explanation of the condition lies in the want of exercise.

It is on the ground of the above experiments and facts in the child's history that I am forced to the conclusion that want of exercise is the chief etiological factor in this unfortunately too common malady. It is possible that there may be a toxin responsible for the immediate results, but without lack of exercise this toxin will not produce any injurious effects. We may surmise that the lack of exercise and fresh air may be productive of a certain amount of perversion of metabolism, which allows of the generation of some harmful product, and so by auto-intoxication brings about the disease. But until this specific toxin is isolated and its nature and mode of formation understood, any such idea is mere theorizing and an easy refuge for ignorance. Typhus fever does not arise *de novo*, and yet we know that somehow or other overcrowding allows the disease to proclaim itself, and by legislation against overcrowding this disease has been kept at bay. Overcrowding and typhus fever are no more closely related to one another than lack of exercise and rickets, and by instilling this fact into the minds of mothers, and especially those of the poorer classes, as well as into corporations who have the welfare of the infant race at heart, rickets would undoubtedly become a very rare disease. Far be it from my intention to belittle dietetics, which is one of the most important branches of pediatrics. Faulty feeding is the cause of much of the infantile mortality, but that it plays any important part in the etiology of rickets is very doubtful.

CONCLUSIONS.

1. Not one of the many theories which have been elaborated to explain the cause of rickets has been universally accepted, and they all lack, not only from the clinical but also from the experimental aspect, unequivocal proof.

2. It is some error in feeding which, in this country and America, is commonly believed to bring about the disease, but it is doubtful, however, if feeding plays any important part in the etiology of rickets. Experimentally I, like several other observers, have been unable to cause the condition by improper feeding.

3. By confining young dogs and depriving them of exercise, rickets has been invariably induced, as in the experiments detailed, and that although their diet was beyond suspicion, the air which they breathed pure, and their kennels were kept scrupulously clean, whereas control animals allowed exercise, but otherwise similarly treated, did not become affected.

4. Examination of the conditions under which rachitic children are reared reveals one constant and invariable factor in their lives, namely, confinement. Alike, then, on clinical and experimental grounds I accordingly conclude that confinement, with consequent lack of exercise, is the main factor in causing the disease.

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DISCUSSION.

Mr. H. GRAY: The paper before us is somewhat difficult for us to discuss, because on the one hand it is not from the pen of the introducer, and on the other the real author is not here to defend it. Be this as it may, Mr. Cousens has taken upon himself the responsibility of agreeing *in toto* with Dr. Finlay's remarks, and no doubt he will, in defending any criticism we may offer upon them, give us some observations he has made from actual experience.

The author discusses the etiological theories of rickets. He considers fœtal rickets is rare and that it should be designated "osteogenesis imperfecta." This form of rickets, however, is very common in the small breeds of dogs, especially the toy spaniels, such as the Japanese, King Charles and its varieties, Pekingese, etc., where the fontanelles remain open during the animal's lifetime; the teeth are few in number, and often the alveolar processes are imperfectly ossified.

He then calls attention to the closely allied theory, congenital rickets, the so-called hereditary theory; that is, the condition is dependent on some inherited weakness or predisposition. I do not think any of us who have had experience with various animals can doubt that there is an inherent predisposition to rickets. I can confidently affirm that this is so, not only in animals but also in the human subject. Hereditary rickets is well seen in the short legged, short nosed animals, where the fontanelles mostly remain open the duration of the animal's life, the teeth are few in number and generally irregularly placed in the dental arcade, the legs are twisted, etc. Hereditary predisposition is also shown in the heavy breed of fowls, in goats, in the pig, etc. It is in fact seen in the heavy lymphatic breeds of animals where the bone is more cancellated than in the fine light breeds where the bone is more dense and compact.

Dr. Finlay then discusses the faulty hygienic conditions, such as incorrect feeding, want of sunlight, etc., generally ascribed by medical men as being the principal factors in the causation of this complaint. To my mind they do play an important part in the production of this malady. When animals are fed well, they become vigorous, are more inclined to frolic and skip about, etc. than half-starved, ill-fed animals kept in dark hovels, which have a depressing influence on the general nutrition of the system. The lactic acid and chronic over-feeding theories we will pass over.

Now we come to the absence or deficiency of lime in the food theory. Dr. Finlay mentions authorities who asserted they could produce curvature of the bones experimentally by depriving animals of earthy salts. This is a theory that has received a good deal of support from veterinary surgeons practising in breeding districts where a disease similar in appearance to, if not identical with rickets is commonly seen affecting lambs. Whether this disease is rickets or not I cannot pretend to say authoritatively, but no doubt some gentlemen in this audience will give the meeting their views.

The next theory is that of Moussu, Professor of Bovine Pathology of the Alfort Veterinary School. It is the infective theory. Moussu was able to convey rickets to young animals by feeding them on the marrow derived from acute cases of rickets ; he could also induce rickets in young pigs by putting them into a shed vacated by rickety pigs. He could not induce the disease by using material from chronic cases. Syphilis has been said to act as a predisposing cause in man. Distemper in the dog is also a predisposing factor in the etiology of rickets. From the description given by Dr. Finlay of his dogs it makes me suspicious that they were not healthy animals before he started his experiments: some had diarrhœa, some died, and one at least died of broncho-pneumonia.

Finally, Dr. Finlay discusses the effects of deprivation of exercise. He says "Lack of exercise as a factor in the pathology of the disease has received remarkably little attention, though to my mind it is of the utmost importance, and in future will always require to be taken into consideration when discussing experimental rickets." I quite agree with this remark, but it is very generally known amongst intelligent farmers, pig breeders, and dog breeders, especially those who breed the larger varieties of dogs, that unless young animals are exercised in the open as early as possible after birth they have a liability to go wrong in their limbs. Years ago bulldog breeders took advantage of this common knowledge and kept young bull pups constantly on the chain so that they should not run about and let their limbs grow too straight, an almost semicircular curve of the limbs in former days being considered by the judges of "the fancy" a point to be sought for. Dr. Finlay says, "By depriving young dogs of exercise I invariably induced the condition, though they received as much fresh air and sunlight as the control animals." He tells us the breeds he experimented with. Now the Collie and Great Dane are two breeds of dog you can easily induce rickets in. The same can be said of several other breeds, but these are more predisposed to it. It is much rarer to find rickets in some of the smaller breeds, however confined. Again, whoever saw rickets in a calf allowed to suckle its mother until it was fit as veal for the butcher? I have hundreds of calves receiving no other food than their mothers' milk and yet confined within a pen about 4 or 5 by 6 ft. for weeks together without the animals developing rickets. Every case of rickets cannot be put down to lack of exercise. It is a fact well known to shepherds that of two lambs of a ewe running out in the open the fat one may be rickety and the lean one normal in limb. Dr. Finlay says marasmic infants never become rachitic. In my experience it is generally the fat heavy child or the clumsy animal that gets the complaint. Would not the weight have something to do with the malady?

Going back to the feeding, Dr. Finlay says however indifferent the feeding was, his dogs never suffered from rickets so long as he gave them exercise. I will give a case that will seem to negative the conclusion that feeding has nothing to do with rickets For many years I have bred budgerigars—the small Australian grass parrakeet. These birds frequently breed during the winter months, when the nights are long. In consequence of the long nights the young do not get fed so many hours as they would in the normal breeding season when the nights are very short and the days long. As a result of the insufficient supply of food the young birds are confined longer to their nest because they have not developed at the proper rate. They therefore become rickety as a sequence to their too long sojourn in the nest. Is this not a fact that goes to show that an insufficient food supply has a place in the etiology of rickets? You can, however, also often induce rickets by taking young budgerigars from the nest and clipping their wings so that they cannot fly.

Another theory, not mentioned by the author, is that rickets is caused by housing dogs and other carnivorous animals, and poultry, on boards. It is a very common condition in dogs so housed, more so when the floor is slippery or polished. Toy spaniels frequently suffer from rickets even though they get plenty of galloping exercise in the house without fresh air. An insufficient food supply, of course, minus exercise in the open, does, I believe, play a contributory part in the etiology of many cases of rickets.

Dr. Finlay (page 149) says, "It is a disease which is found chiefly among children of urban populations, being comparatively rare in the country." Now I do not wish to pose as a medical authority, but think I may claim to give an opinion on facts observed. For nearly thirty years I have had experience of the conditions under which children live, first in the East of London and then in the West. I had before this period and during the interval some experience in the country. Now what has struck me very much is this: that you see more rickets in children in the country than in town. The other Sunday I went into Kent and I saw more cases of rickets in children in that one day than I have observed in London in a twelvemonth. Probably the reason some observers consider it a commoner disease in town than in the country is because they have millions in town to draw their observations from, whereas in the country over the same area they have only thousands, and perhaps not so many. They do not often seem to take conditions into consideration in formulating statistics. Dr. Finlay also says, "Further, it is among the children of the poor and the working classes that we find the disease most frequently, and not among those of the rich." It is not because there are more poor than rich, but the poor frequent hospitals more than the rich; the rich have a family medical attendant who does not always send for the specialist when he has a case of rickets. If it be a rare disease in the rich man's child is it not so because he is better fed and has more change of air and food? I am of opinion the poor man's child gets more exercise in the open than the rich man's child, who is generally confined to a perambulator, go-cart, or nursery. The poor man's child, who is through necessity generally much more precocious than the rich man's child, seems to run about the street as soon has he has left his mother's breast, to annoy or frighten drivers of vehicles or perhaps to get killed. Any cause that leads up to the deprivation of exercise should be taken into account in the etiology of rickets. Dr. Finlay again says, "I cannot believe that the poor in the town feed their children so much less intelligently than those in the country as to account for the great preponderance of the disease among their offspring." My observations go to show that although the country child is coarser fed he is more suitably fed for his constitution than the town child. Dr. Finlay tells us that it is a disease of the temperate zone, being very rare, and in fact practically unknown, in tropical and sub-tropical countries. But he is silent upon the question of children in arctic or sub-arctic regions, where they must necessarily be confined in consequence of the low temperature during the greater portion of the year. Probably they are immune, because their staple diet generally consists of a great amount of fat. If his theory be correct, how does he reconcile the following statement with it. He says, "In the majority of cases a child develops rickets before it has learned to walk." From this does he mean us to infer that a child develops the disease before it has arrived at the age at which it should commence to walk? If so, his theory, on his own showing, must fall to the ground.

We must all agree with Dr. Finlay that rickets is an eminently curable disease, but in our patients we must catch them early and apply our remedies before deformity has become too manifest, else they will remain permanent or deformed cripples. The worst kind of deformity in my experience is when the animal walks on the volar aspect of the metacarpal or metatarsal region, like a plantigrade animal. This is more particularly seen in the collie than in any other breed. Cod-liver oil, raw meat, etc., are generally recommended. But our aim is to prevent rickets. We should endeavour to instil in the head of the dog-owner, poultry breeder, that by observing the laws of nature as regards young animals, rickets can be prevented and much loss and suffering avoided. Nature intended the young animal to play, to gambol, frolic or skip about, to get into mischief; it gave him an instinctive desire to do so; it intended it for a purpose, viz., the development of the osseous framework. I consider that where this object of nature is obviated by the ignorant or careless livestock owner the aid of the R.S.P.C.A. should be invoked to teach him his duty towards the dumb creation over which he has dominion. This Royal Society might do a lot towards animal hygiene. The Kennel Club should also see into it, and not allow their judges to give championships to rickety dogs exposed for competition. A Rule I would like to see established by the Kennel Club would be:—*That no animal, unless in perfect health or condition, congenital or acquired, should be allowed to be benched, and if by oversight a dog being subject to disease or bad condition should slip in and gain a prize the prize winner should be disqualified if objection be raised and maintained.* Such action as this by the Kennel Club would go a long way to improve the hygienic conditions under which dogs are kept. And I think an expression of opinion to this effect should go forth from this Association, which should take a more active part in advising authorities than it has done hitherto.

As to the conclusions Dr. Finlay has arrived at, I think they are on the whole very fair, but is he not a little inconsistent when he in one place says lack of exercise is the chief etiological factor and in another adds "and fresh air." He is inclined to the former but seems afraid to omit the latter. After all, whichever theory be correct, it is a matter of nutrition.

I have been discussing infantile rickets, but according to some authorities, especially to J. Bland Sutton, there are two other forms: (*a*) the rickets of adult age (osteo-malacia, osteo-porosis, etc.) and (*b*) the rickets of pregnancy. The rickets of adult age is not so rare in the dog as some may be inclined to think. I have an example with me. It is the head of a six-year-old male fox-terrier. Both jaws and the face are moveable like rubber. I have seen several examples not only in the skull but also limbs, which have become curved as in ordinary rickets.

THE PRESIDENT: In the continued absence of the apparent author, Mr. Cousens, I think it would be very injudicious on our part to continue the discussion. (Hear, hear).

[The discussion of the paper was not proceeded with.]

Mr. Ainsworth Wilson : I desire to propose a resolution that a committee be formed of this Association to investigate the matter of sterility and the possible existence of contagious granular vaginitis in this country, and report to the next Meeting.

Mr. G. A. Banham : I should be very pleased to second that proposition, but there is one thing that strikes me with regard to all investigations of this kind, that we want funds. Where are the funds to come from ? If you can give us any idea of how we are to obtain the means by which this investigation can take place, I shall be pleased to second the motion.

Mr. Ainsworth Wilson : I would suggest that no funds are necessary in this connection. A committee of practical men who are connected with cattle practice could be formed here and now, and they could appoint a chairman and either meet or not meet during the next twelve months The penny postage would form a means of communication between them, and some scheme might be devised by which their investigations could be communicated one to the other. They could work together on certain lines and present a combined report to next year's Meeting. The subject of sterility in cattle is a highly important one, and is becoming of increasing importance. We really know very little about it. It depends on very many causes, of which infection is only one factor, and in my opinion quite an unimportant factor, and if this committee were formed I think good results would actually accrue. The investigation would be carried on in the private practices of veterinary surgeons in different parts of the country who would compare results from time to time.

The President : Unfortunately Mr. Wilson has not found a seconder for his resolution, but apart from that it appears that the investigation contemplated will be restricted almost to investigation by individuals. That has been done once or twice during the career of this Association. For instance, in one case it was done with regard to hæmo-albuminuria ; a number of members made a careful investigation of the cases that occurred within their practice during twelve months and they recorded results, including the post mortem examinations. A similar investigation was carried on some years before that in connection with milk fever. The only communications that took place, I understand, were in the form of correspondence carried on through the post.

Mr. R. Hughes : And there were very little results.

The President : Unless Mr. Wilson can find a seconder for his resolution it must fall.

Mr. A. Levie : To keep it going and just to put the matter in order, I beg to second Mr. Wilson's resolution.

The PRESIDENT: The resolution is: "That a committee of the National Veterinary Association shall be formed to investigate Contagious Granular Vaginitis and Sterility in Cattle, and to report to the next Annual Meeting."

On the motion being put, it was lost.

The PRESIDENT: Do you demand a vote?

Mr. WILSON: No.

Mr. J. CAMERON: I have to move the following resolution: "That we express our grateful appreciation for the high class papers which have been brought before us from year to year, and we take this opportunity of suggesting that an increase in subjects of surgery and especially surgical demonstrations would be very welcome and, we believe, have an attractive influence; we also believe that many members have invented and possess instruments superior to any others of the same kind that are available for purchase, and would therefore suggest that a prize be given for the best new or improved instrument to be put on sale. And as patents are often not remunerative, a small royalty may be added to the price of each instrument or appliance and handed over to our Veterinary Benevolent Funds (same as Cameron's Veterinary Thimble). We propose that these ideas be transmitted to the Provisional Committee for next year for their sympathetic consideration and approval." It binds us to nothing, and might help us.

The PRESIDENT: There is nothing more required except to read it.

Mr. CAMERON: The other one is: "We, the Members of the National Veterinary Association, now in Annual Meeting assembled, take this opportunity of pointing out to His Majesty's Government the great need there is for further scientific investigation into many diseases of plant and animal life. Also that said investigations cannot be undertaken and brought to a successful issue without the assistance of Government. We therefore respectfully solicit your sympathetic consideration and support thereof. This resolution to be forwarded to Earl Carrington."

The PRESIDENT: The first subject is quite in order, and all that is necessary is that it shall be inscribed upon the Proceedings and appear in the published volume. It is unnecessary to have a seconder, and no discussion is allowed. The second subject unfortunately cannot be accepted without amendment. The concluding clause asks that it be forwarded to Earl Carrington. That involves action, and no action of the Association can take place without due discussion and consideration. Therefore, if you wish this minor subject to be inscribed in the Proceedings, you must withdraw the last sentence or else give notice to the Provisional Committee before the next Annual Meeting.

Mr. J. CAMERON : Then I prefer to delete the last portion.

The PRESIDENT : And have the remainder inscribed on the Proceedings ?

Mr. CAMERON : Yes.

The PRESIDENT : Are there any other resolutions bearing upon the papers discussed ?

VOTES OF THANKS.

Mr. M. HEDLEY : At this part of the proceedings there are always certain important steps to be taken in connection with the Annual Meeting, and I have been asked by the Secretary to be good enough to take the first step, a step I regard as one of the most important, and that is, to propose that the best thanks of this meeting be accorded to our President for the kindly manner in which he has kept us in order, leaving us to go back home to our various residences in different parts of the country with a clear memory of him and kindly feelings towards him (Hear, hear). The duties of the President I need not enlarge upon. Those who have attended many of our meetings know very well what they are. Still there is a good deal of other work to do that we do not see, and I am sure that Prof. Macqueen during the course of his year of office had had a large amount to do. His hands have been full of other duties as well as the duties connected with the Association. I congratulate him, and I am sure we all congratulate him, on the success which has attended his efforts (Cheers).

The resolution was carried with acclamation.

The PRESIDENT : I am sincerely thankful to you, Mr. Hedley, for you kind words, and to the members for the way in which they have responded to the proposal. Since I have had the honour to be appointed as your President for the year which is about to close, I have had extremely little to do, thanks to the energy of our friend Mr. Hunting, who seems to run the National largely as a private concern (Laughter). I believe he has been ably seconded by Mr. Walter Harrison (Hear, hear). Between the two they have carried on everything in the most satisfactory manner as far as I am concerned, and relieved me of all anxiety.

Mr. R. HUGHES : A very pleasant duty has fallen into my hands, namely, to move that we accord a very hearty vote of thanks to the writers of the papers. Writing papers for the National is no small labour; it involves a great deal of preparation and a vast amount of burning of midnight oil. For the writers to tell us all that is known on the subject on which they write is a task of very considerable importance.

The resolution was carried unanimously.

Mr. AINSWORTH WILSON, in replying for the Essayists, said: I thank you very heartily on behalf of myself, and others who are not present, for the manner in which you have carried the resolution. I may say that it does involve, as Mr. Hughes says, a burning of the midnight oil, but I think we have met with a very good reception at your hands, and I am sure we are only too pleased to do what we can. I take it each of us has written about something we are interested in, and it is to our own benefit as well as to the benefit of others, if there is any benefit at all connected with it.

Col. C. STEEL: I have pleasure in proposing that the thanks of the meeting be accorded to those gentlemen who have opened the discussions. I have been very much struck with the ability and impartiality with which the papers have been criticised. The criticism has sometimes been rather severe perhaps, but so much the better for opening up the field for discussion. The best proof of the discussions having been ably opened is the excellent discussions that followed on the openers' remarks.

The resolution was carried unanimously.

Prof. WOOLDRIDGE: I thank you for your reception of the motion, and will leave Mr. Gray to say the rest.

Mr. H. GRAY: I beg to thank you on behalf of the openers of the discussions and on my own behalf for the manner in which you have received the resolution. My colleagues have had a much more difficult task than I have had: their criticisms have been answered and mine have not.

Mr. S. VILLAR: When I had the honour of addressing a few words to the meeting yesterday morning I suggested that the Provisional Committee was to be congratulated on the choice of the subject we were then discussing, the subject of stringhalt and shivering in the horse. I think you will agree with me that not only are they to be congratulated upon the whole of the subjects which they have selected for your discussion at this meeting of the National Veterinary Association. That they have been subjects full of interest is shown by the prolonged discussions that have taken place on each of the papers. But the selection of subjects is a very small part of the work which the Provisional Committee is called upon to perform and I am sure you would wish to thank them for all the hard work, the trouble, and the time, they have spent over this matter (Hear, hear). I would particularly allude to the services which have been rendered by the Chairman of the Provisional Committee, Mr. Salusbury Price (Hear, hear), and by his energetic Secretary and helper, Mr. Harrison (Hear, hear). I have met Mr. Harrison a good deal in the course of the preparations for this meeting and

I never saw a man who worked more energetically and with greater love for his profession than he has.

The resolution was carried with acclamation.

Mr. T. Salusbury Price: As far as the Provisional Committee is concerned I return you many thanks. I can assure you that what we have done has been out of love for the profession. I can fully bear out what has been said about Mr Harrison, whose help and assistance have been very great, and also I should like to refer to the assistance we have had from Mr. McCormack.

Mr. C. Roberts: I propose with a great amount of solid satisfaction a vote of thanks to the Officers of the Association, particularly to the Secretary and Treasurer. I happen to know something of the great work that has to be done by those officials throughout the whole of the year. Their work does not begin and end with the two days, as with us. With them it is a matter of solid work throughout the year in organising the details of the meetings. I know a good deal of it, but I know that there is a great deal more of which I am ignorant.

The resolution was carried with acclamation.

Mr. Hunting: I thank you for the vote of thanks, though I must confess I do not deserve it. Mr. Harrison has done all the work—and Professor Macqueen (laughter). Some years of experience have shown me that the only possible way in which a General Secretary can run a show of this kind is to let every man do as he likes, and that is what I endeavour to do (laughter).

Prof. Wooldridge: I only took up the work of the Treasurer during the past year, in the middle of the year, when Mr. Simpson was unfortunately unable to carry it on. That you are satisfied to some extent with my effort is shown by the fact that you have re-elected me for the coming year. I will certainly do the best I possibly can for the Association (Hear, hear). I heard some of the previous officers making conditions the other day, and I also must make one condition, namely, that the members of the Association assist me. I do not think it is at all fair to ask the Treasurer to keep writing to various members to remind them that their subscriptions are due. Subscriptions fall due at the beginning of the year and those which have not been paid this year are seven months behindhand. Some of you are exceedingly good in sending your cheques almost by return when reminded, but others put the paper on one side, thinking it would be all right on another day. I hope you will assist me by sending your subscriptions along at an early date (Hear, hear).

The President: That concludes the business of this meeting, Gentlemen, and I thank you very much for your attendance, for your consideration, and for your kindness.

The meeting then concluded.

LIST OF MEMBERS OF THE NATIONAL VETERINARY ASSOCIATION, 1910.

Those marked (*) are Life Vice-Presidents.
,, ,, (1) have served as President.
,, ,, (2) ,, as Vice-President.
,, ,, (3) ,, on the Council.
,, ,, (4) ,, as Treasurer.
,, ,, (5) ,, as General Secretary.
,, ,, (6) ,, on Provisional Committee.
,, ,, (7) ,, as Local Secretary.
,, ,, (8) have written papers for discussions.
,, ,, (9) ,, opened discussions.
(F) signifies Fellow of the Royal Coll. Vet. Surgeons.

Abson, J., (F) (2, 3, 6, 7) 53 Norfolk St, Sheffield
Ackroyd, W., Halifax
Aitken, John, Junr., Dalkeith
Allen, Chas., (F) (* 1, 2) Dublin
Almond, N., (F) (8) Cambridge House. Kingston-on-Thames
Anderton, F. W., 59 Manning Lane, Bradford
Archer, A. H., 1 Stafford Rd, Southsea
Ascott, Wm., (6) Bideford
Auger, A. S,, (6, 9) Saxmundham
Auger, T. E., (6) Wymondham, Norfolk
Awde, W., (F) (2,3,6) Stockton-on-Tees
Baird, A., York Lane, Edinburgh
Baker, E.W., (2) Wimborne, Dorset
Banham, Geo. A., (F) (2, 3, 5, 6, 7, 8) 32 Rock Road, Cambridge
Banham, G. F., Cambridge
Barber, G. G., Long Close Lane, Leeds
Barcham, T. E., Paston, North Walsham
Barling, F. W., (3, 6) New House, Ross, Herefordshire
Barron, R., Blandford
Batt, T., (6) 469 Oxford St, London
Bayley, A. E., 144 Lordship Road, N.
Beart, W. J., King's Lynn, Norfolk
Beckett, A. J., (6) 3 Marlboro' Road, St. Albans
Beddard, E., (F) (2, 3) Cleveland Road, Wolverhampton
Bell, G. J., 2 High Street, Wandsworth
Bell, John J,, (2, 3, 6) Carlisle
Bett, R. F., A.V.C., The Gables, Aldershot
Blake, A., (F) Rangoon, Burma
Blakeway, Jno., (F) (6, 7) Chaddesley Corbet, near Kidderminster
Blench, J., Birdgate, Pickering
Blenkinsop, Col. L. J., A.V.S.
Bloxsome, Gerald W., (6) 15 Davigdor Road, Hove
Bloye, W. H., (F) (* 1, 2, 3) Ebrington Street, Plymouth
Bond, P. G., (6, 7) Plymouth
Borthwick, Jas., Gateside, Kirkliston
Bowden, A. Cornish, 127 High Street, Beckenham
Bower, Wm, (* 1) East Rudham. Norfolk
Bowes, H. G., (F) (6) 106 Clarendon Road, Leeds
Bowman, G. E., (* 1, 2, 6) Woodhouse Lane, Leeds
Bradley, O. C., (7, 8) Veterinary Coll., Edinburgh
Branford, Richard, I.C.V.D., c/o King and Co., Bombay
Broad, Alf., (F) (3,6) 8 Finchley Road, St. John's Wood, London

Broad, Arthur, 53 Elsham Road, London, W.
Brooke, W. H., (6) Handsworth, Birmingham
Brooks, H., Leadenham, Lincoln
Burchnall, J. J., (6) Loughborough
Burrell, T., (6) 50A Brewer Street, Regent Street, London
Burt, Walter, Junr. (F) (2,6,7) 2 Edward Street, Brighton
Butler, E.R.C., (F) Lt.-Col. A.V.C., (3, 9) Simla, India
Butters, A. L., (3) 126 Ledbury Rd., Bayswater, W.
Byner, C. J., 71 Broad Street, Birmingham
Cameron, J., Bridge Street, Berwick
Carless, W., (2, 3, 6) Eastgate Street, Stafford
Carless, W. S., (2, 3) Worcester
Carr, F. U., Capt. A.V.C., Aldershot
Carter, J. H., (F) (2, 3, 6) Burnley
Carter, J. T. Potter, Endymion, Homefield Road, Gt. Malvern
Caton, H. W., (F) (6) 413 Mile End Road, London, E.
Caudwell, W., (F) 20 London Street, Chertsey, Surrey
Challinor, C. E., (F) (6) Pendlebury
Chase, W. H., (F) Mafeking, S. Africa
Clarke, W. R., 18 Gayton Road, Hampstead
Clarkson, J., (6, 7) Garforth, Leeds
Clough, F. W. S., (6) 4 Zoar Street, Morley, nr Leeds
Cockburn, R., (6) Eastwood, Notts.
Coe, J. W., (F) (3, 6, 7) Stoke-on-Trent, Staffs.
Connell, J. A., Lichfield
Connochie, T. D., Galashiels
Constable, D,, (3, 6), Inchture, Perthshire
Craig, Prof. J. F., M.A., Royal Veterinary College, Dublin
Crawford, W., (6) Woodhouse Lane, Leeds
Creagh, P. W., Fermoy, Ireland
Crowhurst, E. G., (F) (3) Hindley House, Leamington
Cunningham, C., (2, 3) Slateford, Midlothian
Daly, J. V., (3) Bo-Peep, Island Bridge, Dublin
Darwell, A. H., (3, 6) Northwich, Cheshire
Davies, D. G., (2, 3) Heathfield, Swansea
Dawes, H. J., (F) West Bromwich
Dawson, W., (6) Cavan
DellaGana, W. A., (F) Above Bar, Southampton
Dewar, J. R. U, (F) (* 1, 8, 9) Prin. R.V.C., Clyde Street, Edin.
Dixson, E. L., 11 Grosvenor Place, Margate
Dobbyn, A., (2, 3, 6) Waterford
Dollar, Jno. A. W., (6, 8) 56 New Bond Street, London
Douglas, T. A., (6) Kilmarnock
Duck, F., (F) C.B., Col. A.V.D. 35 The Avenue, Surbiton
Duckworth, T. H. L., Ashbourne, Derbyshire
Dudgeon, G. R., (3, 6) Sunderland
Dunlop, J. B., (6) Ailesbury Rd, Dublin
Dunne, G., (F) 102 Upper George St, Kingstown, Co. Dublin
Dunstan, J., Prof. (3, 6, 8) Liskeard
Edmonson, W., (6) Wansford House, East Parade, Harrogate
Edwards, E. R., (F) Bd. of Agriculture, 4 Whitehall Place, London, S.W.
Edwards, F. G., (F) (3, 6) Chester
Edwards, W. T., Windsor Rd., Neath
Elder, G. H., Taunton
Elder, W. A., G.V.S, Mbabane, Swaziland, S. Africa
Ellison, A., (6) Harrogate
Elphick, G., (2, 3, 6) 67 Newgate St, Newcastle-on-Tyne
Faulkner, Ed. (* 1, 3, 6, 8) Knott Mill, Manchester
Fletcher, T. C., (6) Ellin St, Sheffield
Foreman, R. J., 401 High Rd, Tottenham
Foreman, W. J,, Leadgate, Co. Durham
Forsyth, A. B.. Cannock
Freeman, R. B., (2,6) Harry St, Dublin
Garnett, F. W., J.P., (* 1, 3, 9) Windermere
Gibbings, G. H. (F) (3, 6) Tavistock
Gofton, Prof. A., R. V. Coll., Edin.
Golledge, C. H., Sherborne, Dorset
Gooch. F. L., (F) (2, 3, 6) St. Martin's, Stamford
Gooch, J. K., Holt, Norfolk
Goodall, T. B., (F) (2, 9) Christchurch, Hants
Gosling, J. A., (F) (6) 212 Goswell Road, E.C.
Grasby, W., (2, 6) Daventry
Gray, H., (3, 6, 8, 9) 23 Upper Phillimore Place, Kensington, W.
Green, Herbert, (6) 26 Hampshire Terrace, Southsea
Hall, R. W., Tynewydd Road, Barry Dock
Hallilay, F., (6) Southfield House, Wakefield Road, Dewsbury

Hamilton, D., (F) Bourtreehill, Hamilton
Hamilton, T. B., 183 Queen's Drive, Strathbungo, Glasgow
Hamilton. William, Cold Bath Road, Harrogate
Hammond, John, (2) Bale, East Dereham, Norfolk
Hancock, W. A., (3,6) Clowden House, Uxbridge
Harding, E. R., Salisbury
Hartley, Chas., (F) (2, 3, 6) Lincoln
Haywood. Chas., Millstone Lane, Leicester
Healy, J. F., (2, 6) Midleton, Co. Cork
Heatley, T. G., (6) Woodbridge
Hedley, M., (F) (*1, 2, 3, 7) 6 Royal Terrace, Kingstown, Dublin
Heelis, L. W., Solihull
Hewson, Frederick (6) Royston, Herts
Hickes, R. J., (F) (6) Market Weighton
Hill, J. Campbell, Town Hall, Llanelly
Hoare, E. W., (F) (2, 6, 8) 18 Cook Street, Cork
Hobbs, F. C., 105 Cærleon Road, Newport
Hobday, F., (F) (3, 7, 8, 9)
165 Church St, Kensington, W.
Hodgman, J. A., (6) Barnsley
Holburn, A., (6) Congleton, Cheshire
Holland, J., (6) Athy, Co. Kildare
Hopkin, Tedbar, (F) (2, 3, 6, 9)
15 New Bridge St, Strangeways, Manchester
Houston, W. F., (3) Paisley
Howard, C. W., 43 High St, Dorking
Howard, P. J., (2,3,8) Ennis, Co. Clare
Howard, P. S., (F) "Windermere," Cambridge Park, Wanstead, Essex
Howe, G., (F) (3, 6) Buxton
Howes, W. F,, (6)
Hughes, R., (F) (2,3,6) Willow Street, Oswestry
Hunter, H., (2, 3, 6, 9) New Bridge Street, Newcastle-on-Tyne
Hunting, C. S., South Hetton, Durham
Hunting, W., (F) (*1, 3, 5, 6, 8, 9)
5 Halkin Place, Belgrave Sq, S.W.
Hurndall, J. S., (2, 3, 6, 8, 9) 2 Cornwall Gardens Stables, Kensington
Inglis, T. M., Forfar
James, J. C., (F) Thornbury, Glo'ster
James, W. H., Frome
Jermyn, W. R., Clifton, Bristol
Jones, C , (6) Vet. Dept. Dublin
Jones, J. T. Share, (F) Liverpool University
Jones, R., Towyn, Merionethshire
Jones, T. Eaton (6,9) Municipal Bdgs., Liverpool
Jurgenson, W. M., (6) Ipswich
Kendall, W., (F) Buccleuch Street, Barrow-in-Furness
Kerr, F., (2, 3, 6, 7) Vet. Dept, Dublin
Kidd, H., (F) (2, 3, 6, 8) Exe View, Exmouth
King, H., (6) 155 Lower Kennington Lane. S.E.
Laithwood, James, (F) (3) Congleton
Lambert, R. H., (6) South Richmond Street, Dublin
Lambert, T. D., (F) (2, 3, 6) 47 South Richmond Street, Dublin
Lawson, A., Manchester
Leach, E. H , (F) (2, 6) Newmarket
Lepper, H. G,, (3) Aylesbury
Levie, A., (F) Broad St. Nottingham
Livesey, Geoffrey H., (6, 8)
87 St. Aubyns, Hove
Lloyd, J. S., (F) D.V.S.M. Vict.
(2, 6, 8, 9) Town Hall, Sheffield
Locke, G. H , Grosvenor St, Manchester
Lomas, S., Wilmslow, Manchester
Lowe, G. C., (F) Oxley Street, Dockhead, S. E.
Macarthur, J., (3) Southminster, Essex
McCall, J., (F) (*1, 2, 3, 8, 9)
Principal, Vet. College, Glasgow
McCall, James McI., (3) Board of Agriculture, 4 Whitehall Place, S W.
McCall, J. R., (8) Buccleuch Street, Glasgow
MacCallum, A. I., (3, 6) 30 Kings Stables Road, Edinburgh
McCarmick, A., (6), Kirkstall Road, Leeds
MacCormack, H. A., (6) 122 St. George's Avenue, Tufnell Park, London, N.
M'Fadyean, Sir John (* 1, 2, 3, 8, 9), Principal, R.V.C. London
Macfarlane, D., (3) Doune, Stirling
McGavin, James, (2,3,9) Montgomery
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If any errors occur in the above list, please communicate with the General Secretary, W. Hunting, 5 Halkin Place, Belgrave Square, S.W.